A CASE-CONTROL STUDY OF HOUSEHOLD AIR POLLUTION AND TUBERCULOSIS IN WOMEN AND YOUNG CHILDREN IN URBAN INDIA

by
Jessica L Elf, MPH

A dissertation submitted to Johns Hopkins University in conformity with the requirements for the degree of Doctor of Philosophy

Baltimore, Maryland
December 2015

ABSTRACT

Background: Household air pollution (HAP) is a known risk factor for many respiratory diseases, however the association between HAP and tuberculosis (TB) is still inconclusive. The purpose of this study is to characterize exposure to common sources of HAP in low-income urban Indian households and estimate the association between HAP and TB.

Methods: A matched case-control study was conducted among adult women and children. Index cases were recruited from TB control program clinics, and controls were healthy individuals matched on geography, age, and sex. Particulate matter less than 2.5 microns ($PM_{2.5}$) and carbon monoxide (CO) concentrations were measured in each household and compared across case and control participants. Household use of cooking and heating fuels, as well as reported exposure to other sources of HAP was collected. Exposure to secondhand tobacco smoke (SHS) was measured through household air nicotine concentration and participant hair nicotine concentration.

Results: A total of 127 households were enrolled, 8 of which were excluded due to incomplete household exposure data. In the case-control analysis, 118 individuals with complete case-control pairs were included. Kerosene was used by 26 (22%) of households, and 32 (27%) of households reported using wood as a primary or secondary fuel source. The median 24-hour mean concentration of $PM_{2.5}$ in households was 184 $\mu g/m^3$ (IQR: 113-347), well above the recommended 24-hour guideline of 25 $\mu g/m^3$. Odds of TB was marginally statistically significantly associated with higher levels of $PM_{2.5}$ (OR 3.30, 95% CI: 0.95 - 11.51). Those in the upper quartile of exposure were more likely to use wood (OR 5.53; 95% CI: 1.62 - 20.39) and mosquito coils (OR 3.82; 95% CI: 1.31 - 11.79). Reported measures of exposure to SHS were not valid in this population, weakly identifying those with detectable air (n=31, 32%) and hair (n=42, 68%) nicotine.

Discussion: Households in low-income urban communities are highly exposed to HAP from cooking fuels, SHS, and other pollutant sources. High levels of HAP exposure tend to be associated with TB, raising concern for implications at the population level in countries with dual burden of both HAP and TB.

DISSERTATION ADVISORS AND READERS

Jonathan Golub, PhD, MPH

Associate Professor, Primary Thesis Advisor and Dissertation Reader

School of Medicine

Patrick Breysse, PhD

Professor, Thesis Advisor and Chair of the Committee

Department of Environmental Health Sciences, School of Public Health

Amita Gupta, MD, MHS

Associate Professor, Thesis Advisor and Dissertation Reader

School of Medicine

Andrea Ruff, MD

Associate Professor, Dissertation Reader

Department of International Health, School of Public Health

Luke Mullany, PhD, MHS

Professor, Dissertation Reader (Alternate)

Department of International Health, School of Public Health

Colleen Hanrahan, PhD

Assistant Scientist, Dissertation Reader (Alternate)

Department of Epidemiology, School of Public Health

ACKNOWLEDGEMENTS

First and foremost, this project would not be possible without the generosity of the study participants and their families who volunteered their time and opened their homes to us. They afford us the opportunity to advance science, and our own careers, without direct equivalent benefit in return, and I will strive to use the products of this research in ways that honor their immense contribution. Secondly, our dedicated field staff, Sunita Patekar and Vaishali Kulkarni, have worked incredibly hard and are the foundation of the data collection process. Their care for the communities they serve is admirable and inspiring, and I am lucky to count them as colleagues and now friends.

I would also like to acknowledge our collaborators at BJMC/SGH, who have been integral in the scientific and logistic success of this project. I am proud to have the opportunity to work with you, and admire the care you provide to some of the most vulnerable in your community. I would like to thank the staff of the CTU for their help with project implementation, and to the India-based JHU faculty, Vidya Mave, Nishi Suryavanshi, and Nikhil Gupte, for their critical guidance throughout my time living and working in India. Importantly, huge thanks to Jyoti Mathad and Andrea Deluca for your friendship, housing, and helping me through.

Thank you to my thesis committee, Patrick Breysse, Jonathan Golub, and Amita Gupta. You have spoiled me with your confidence, encouragement, and guidance, and I am fortunate for having the opportunity to work with each of you. Jon, as my primary advisor (and sometimes mental health counselor), you have been an incredible role model for me as a mentor, educator, and academic. I truly value your honesty, your kindness, and your friendship.

I would like to acknowledge my amazing family and friends, who live both near and far. To have the support of such inspirational, incredible individuals is beyond words. Lindsey Horton, Jessica Carda-Auten, Jessica Gehle, Laura Grossmann, and Amber Robinson, you are intelligent, strong, beautiful women who inspire and motivate me, and have given me the courage to try even when I doubted myself. Thank you to my parents, Paula and Bryon, and my sister, Lindsey, for supporting me throughout the "anything" it was I wanted to do. I hope you are proud.

Finally, all of my gratitude to Leanna Berger, for holding my hand throughout this process and putting up with all of the madness. Thank you for all of your support, long-distance conversation, and rendezvouses in fabulous places. I couldn't have done it without you.

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CHAPTER 1

Introduction

Since declaring tuberculosis (TB) a public health emergency in 1993, great strides have been taken by the World Health Organization (WHO) and their partners to curb the epidemic in countries throughout the world. Directly Observed Treatment - Short Course (DOTS) was launched as the WHO-recommended strategy for government control programs, consisting of political commitment, sputum smear microscopy for diagnosis, a stable supply of first-line anti-TB drugs, short-course chemotherapy, and standardized recording and reporting of TB cases and their outcome to the WHO[1]. In 2000, TB was incorporated into the Millennium Development Goals for 2015, with Target 6c being to halt and begin to reverse the incidence of TB[2]. The Stop TB Partnership, an international collective hosted by the WHO, was established in 2001, and developed the Global Plan to Stop TB, which established targets for reductions in disease burden as compared to a 1990 baseline[3]. By 2015, targets were to reduce both worldwide prevalence and death rates of TB by 50%[4].

With these global partnerships, great progress has been made to reduce the burden of TB worldwide. Among countries that have adopted the recommended WHO strategies, approximately 66 million people have been successfully treated for TB in the past 20 years, and 43 million lives were saved between 2000 and 2014[5]. Globally, the MDG target of halting and reversing TB by 2015 has been achieved, though the global decline is minimal, and TB remains a significant cause of morbidity and mortality worldwide. There were an estimated 9.6 million new cases of TB in 2014 and 1.5 million deaths in individuals with and without HIV infection[5]. Post-2015, the WHO is promoting the End TB Strategy to end the global TB epidemic, with 2035 targets of reducing the number of deaths by 95% as compared to 2015 as well as to decrease incidence by 90%[5]. The majority of the TB disease burden is shouldered by 22 countries, which account for over 80% of total worldwide cases of TB. The highest burden of disease is found in Asia and Africa, and India and China alone account for nearly 35% of TB cases worldwide.

Although HIV is often the most concerning risk factor for TB disease, 88% of the estimated new cases and nearly 75% of TB deaths in 2014 were in HIV-uninfected individuals[5].

Pediatric TB accounted for approximately 1 million of the incident TB cases and 140,000 deaths in 2014. Childhood TB is an often overlooked and understudied disease, as adults with pulmonary TB often garner the most attention due to their infectiousness. In the 22 high-burden countries (HBC), approximately 95% of incident childhood TB cases are in HIV-uninfected children, and only 35% of pediatric cases were detected, highlighting the importance of prevention in this population[6]. Additional strategies for prevention and control are especially important among children, a population too often neglected in research and one in which risk factors are poorly understood. Children with TB do not present with traditional symptoms, and often have more severe forms of TB and extra-pulmonary TB, as compared to adults. Diagnosis is also extremely challenging, which contributes to the lack of understanding of risk factors for disease, often requiring a tiered scale of diagnosis including confirmed, probable, and possible (or clinical) diagnosis based on microbiologic confirmation, clinical symptoms, known exposure, and failure to respond to non-TB drug treatment regimens[7]. Further, children are not considered to be as highly infectious as adults, and therefore do not command the same attention for prevention or diagnosis as adult populations. These characteristics of TB in children lead to the marginalization of childhood TB in TB control programs and research, and a lack of understanding on control strategies in this population[8].

With a population of over 1.2 billion people, India has the largest number of annual incident TB cases in the world, with an estimated 2.2 million in 2014 and 220,000 TB-related deaths, representing 23% of the global total of incident disease. Only 110,000 of incident TB cases were HIV-co-infected, representing 5% of incident cases. It is estimated that 4-21% of TB cases in

endemic areas are among children, although estimation is difficult due to diagnostic challenges as well as poor systems of surveillance and reporting[6, 8]. India has 27% of the total pediatric burden of TB among the HBCs[6]. With an annual rate of infection of 1.0%, TB infection and subsequent disease remains a major public health threat in India, especially to those in poor socioeconomic conditions.[9]

Social Determinants of Health

Traditionally, the focus of TB control programs has been on detecting and successfully treating active cases of disease, and providing isoniazid preventive therapy (IPT) to children and individuals with HIV infection who are contacts of active cases.[27,31] However, public health professionals have begun to highlight the importance of controlling risk factors for disease as a component of a successful TB control program.[32] TB is heavily influenced by social, economic, and environmental conditions, and individuals in lower socioeconomic conditions are generally at higher risk for disease. Broadening the strategy of TB control to include reduction in social and other health risk factors has recently been promoted as a potentially viable way to complement ongoing case-finding and medical intervention strategies.[2] It has been hypothesized that these types of supplemental interventions may benefit TB programs by decreasing infection, decreasing progression to active disease among those with latent infection, as well as improve treatment outcomes in patients undergoing anti-TB chemotherapy.[32]

The mechanism of association of social determinants of health to increased risk for TB is likely through a decrease in immune function, resulting in a higher susceptibility to TB disease and negative TB outcomes. Through suppression of the immune system, individuals are more likely to acquire TB infection and progress to active disease[10]. Although the degree of association is

clearly hypothesized to be much less for social determinants risk factors as compared to HIV, reductions in these risk factors, or case-finding based upon risk profile, may lead to greater reductions in overall TB burden due to large population prevalence of exposure[11]. Social determinant risk factors may be at the individual, household, and/or community levels, contributing with either an independent effect or potentially having an interaction with one or more other risk factors. Each also has a varying degree of evidence to support its contribution to disease.[32] Once the degree of association, prevalence and importance of these risk factors in communities are better understood, targeted interventions may be used to help decrease those risk factors that are modifiable and aid TB control programs in their control efforts.

Household Air Pollution

Despite being associated with other respiratory disease and having a strong biological plausibility for increased risk, household air pollution (HAP) has not yet been definitively identified as a risk factor for TB infection or disease[12]. HAP typically refers to the combustion products created from cooking and heating fuels burned in the home. In low and middle-income countries (LMIC), this type of air pollution represents the majority of pollution exposure, as it is concentrated in a smaller volume area where a higher dose is acquired, as compared to outdoor air pollution. Fuels that are typically burned include wood, animal dung, crop residues, and grasses, and they emit high amounts of pollution with a relatively low amount of efficiency, as compared to other fuels such as liquid petroleum gas (LPG)[13]. Products of combustion of these types of fuels may include damaging compounds such as particulates, carbon monoxide (CO), nitrogen oxides, formaldehyde, and polycyclic aromatic hydrocarbons (PAHs)[14]. Globally, exposure to HAP is estimated to be responsible for more than 3.5 million annual deaths from respiratory, cardiovascular, and other disease[15]. The current evidence base is suggestive of an association

between HAP and TB, however limitations in study design and exposure assessment have prevented more definitive conclusions[12].

Several recent systematic have summarized the current body of literature assessing the association between HAP and TB. Lin et al. reported on 15 studies investigating HAP from biomass sources and TB in both adults and children, comparing exposure from these sources to "clean fuels", which included LPG and biogas. A summary odds ratio of 1.17 (95% CI: 0.83 – 1.65) was reported for inclusion of all populations, however when restricted to females the estimate changed to 1.63 (95% CI: 0.74 – 3.57)[12]. While the effect size increased, no statistically significant association was found, reportedly due to low quality of many of the existing studies. Outcome measures of TB disease were often strong, but measurement of exposure to HAP was limited to reported exposure to HAP using fuel type and cooking patterns as a proxy for exposure. This assessment strategy is subject to recall bias and may lead to misclassification of exposure. No objective measurements, such as biological markers or environmental markers, were used in any of the included studies.

One of the more recent and stronger case-control studies included in the systematic review was conducted among women in northern India and reported by Lakshmi et al. 2012. While this is not the first study reported from India, many other studies have either failed to control for important confounding factors such as SES or improperly measured exposure and/or outcome[16-18]. Details regarding the type of fuel, kitchen characteristics, and duration of exposure were also not included in most other studies. Lakshmi et al. collected more detailed information on exposure that was missing in these previous studies. Kitchen locations were noted, as was specific type of fuel used, how long cooking was done each day, and lifetime exposure to HAP. Other potential confounding factors, such as socioeconomic status (SES), exposure to secondhand tobacco smoke

(SHS), and crowding were appropriately taken into consideration, and an adjusted OR of 3.14 (95% CI: 1.15 - 8.56) was reported[19]. This study, with its methodological strengths, adds more support for HAP as a risk factor for TB.

Dual Burden of Disease from TB and HAP

Many high-burden TB countries also suffer from a high burden of exposure to HAP, and both are closely associated with low SES and poverty. Asia and sub-Saharan Africa have the greatest burden of solid fuels, and also the greatest burden of TB. Sub-Saharan Africa has over 20% of the total number of people worldwide relying on solid fuels for cooking and heating. India and China, who also have the greatest number of incident TB cases, make up 27% and 25% of the total relying on biomass fuels, respectively[20]. Understanding the risk associated with exposure to HAP from the use of biomass fuels and SHS will provide needed evidence for developing interventions to prevent morbidity and mortality from TB in settings with high levels of these exposures.

Exposure to Secondhand Tobacco Smoke

Use of smoked tobacco has been definitively linked to respiratory diseases, and current evidence shows an association between cigarette smoking and increased risk for TB infection, disease, and mortality[21]. While association with personal smoking is established, evidence available measuring the risk of TB associated with exposure to SHS indicates a potential positive association, but weaknesses in current studies prevent a definitive conclusion[22]. In their recent systematic review, Patra et al. report major limitations of existing studies to include HAP from biomass sources as a controlling factor. Additionally, SES was poorly controlled for in many of the included studies. After sensitivity analysis adjusting for these variables, SES attenuated the effect size of SHS on TB. Given the close relationship between TB, SES, and HAP, additional well-designed and properly controlled for studies are needed[22].

Importantly, most all of the studies included in the previously mentioned systematic review relied on reported measures of exposure to define exposure groups. Questionnaire methods relying on self-report are routinely used for the collection of information regarding personal tobacco use as well as exposure to SHS. Biochemical or environmental validation of responses is often not feasible, especially in large population surveys, for logistical as well as financial reasons. Additionally, the collection of biological samples, such as blood, urine, and saliva, may be deemed as invasive or culturally insensitive in some situations.[23] However, the validity of reported measurements are an ongoing concern, especially with increased stigma regarding acceptability of tobacco use due to health concerns.[24-26] As tobacco use becomes more of a socially undesirable behavior, it can be expected that accuracy of self-report for use may decrease. This may especially be the case in specific populations where tobacco use may be particularly harmful and seen as especially undesirable. Pregnant women and individuals with a smoking-related condition are two examples of such populations[27, 28]. This is of particular concern as these populations are at increased risk for negative health outcomes related to their tobacco use[29]. In general, socially undesirable behaviors have been found to be routinely underreported, and tobacco use is no exception[29, 30].

Children's exposure to SHS is particularly challenging, as it is most often based on parental report of a child's exposure. These reports may be inaccurate or biased if adults fail to accurately report child exposure because of guilt of exposure to a damaging agent. Additionally, best practice measures for assessing SHS exposure are rarely used in this type of study, potentially introducing bias into the results[21].

Underestimates of personal tobacco use and exposure to SHS have important implications for population-level epidemiology. Misclassification of exposure can lead to bias in risk estimates of SHS-disease relationships or when controlling for SHS as a confounding factor. This importance is heightened in populations of especially high risk or for whom tobacco use may be of particular importance, such as individuals at risk for TB disease. The validation of self-reported measures in these populations is necessary as accuracy of self-report may vary across populations and cultures. Additionally, understanding the expected accuracy of self-report will help to better assess how risk associations with tobacco use and levels of prevalence may be adjusted to develop more accurate estimates. Valid reported measures of exposure are also of importance for clinical settings, and help clinicians accurately identify patients who are in need of intervention [31].

To date, there is limited evidence of validation of reported measures for SHS exposure and personal tobacco use in India[23, 32]. Questions used in the Global Tobacco Surveillance System are validated for understanding and comprehension of questions, but routine validation with biomarkers is not conducted (as is the case with the NHANES survey in the US).[33] Additionally, validation of tobacco and SHS exposure use questions in tuberculosis patients has not been exclusively conducted, and may provide value to clinicians and researchers evaluating tobacco use in patients.

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CHAPTER 2

The association of household air pollution and tuberculosis in women and children in Pune, India

Abstract

Background: Household air pollution (HAP) is a known risk factor for respiratory disease, however has yet to be definitively association with tuberculosis (TB). High quality studies are needed to assess the TB risk associated with exposure to HAP and provide needed evidence for intervention strategies among adults and children in India and other settings with these exposures.

Methods: A matched case-control study was conducted among adult women and children index TB patients and healthy controls matched on geography, age, and sex. Exposure to HAP was assessed using structured questionnaires for cooking fuels and other sources of HAP, and measured concentrations of $PM_{2.5}$, CO, and air nicotine were collected in each household. Exposure to HAP was compared across cases and controls using conditional logistic regression.

Results: A total of 118 individuals in 59 matched pairs were included. High levels of exposure were found across all homes, and exposure metrics for high HAP tended to be associated with TB. The use of kerosene was significantly associated with TB (OR 11.18; 95% CI: 1.24 - 100.8) in adjusted analysis. High concentrations of household pollutants tended to increase odds of TB across all measures. Households with the greatest number of hours of $PM_{2.5}$ above 75 $\mu g/m^3$ had a marginally statistically significantly increased odds of TB (OR 3.30; 95% CI: 0.95 - 11.51), and a dose-response effect was seen with increasing quartile, although this did not reach statistical significance.

Discussion: Measures of HAP approached a statistically significant association with TB in multivariate analysis, indicating a likely increase in odds for TB with exposure. Across all study homes, participants were exposed to extremely high levels of $PM_{2.5}$, regardless of their reported fuel use. Interventions are needed to reduce exposure in Indian populations, with the likely benefit of preventing TB disease in this population burdened both by a high incidence of TB and high exposure to HAP.

Introduction

Despite strides in global tuberculosis (TB) control, TB remains a significant cause of morbidity and mortality worldwide. Although HIV is commonly the most concerning risk factor for TB disease, 88% of the estimated 9.6 million new cases and 73% of the 1.5 million TB deaths in 2014 were in HIV-uninfected individuals[1]. Further, in the 22 high-burden countries (HBC), approximately 95% of incident childhood TB is in HIV-uninfected children, and only 35% of estimated pediatric cases were detected, highlighting the importance of prevention in this population[2]. In India, which shoulders the highest annual burden of incident disease in the world and where the TB epidemic is not driven by HIV, environmental and social risk factors are likely the primary contributors to the persisting epidemic[1, 3]. These risk factors may be especially important among children, a population too often neglected in research, and one in which risk factors are poorly understood. It's estimated that 4-21% of TB cases in endemic areas are among children, although estimation is difficult due to diagnostic challenges as well as poor systems of surveillance and reporting[2, 4].

Household air pollution (HAP) is a known cause of lung disease, including asthma, acute lower respiratory infection (ALRI), and chronic obstructive pulmonary disease (COPD), primarily affecting those living in low- and middle-income countries (LMIC)[5, 6]. Generated largely from household use of biomass fuel, kerosene, and secondhand tobacco smoke (SHS), HAP is suspected to increase risk of TB, however current evidence is inconclusive[7, 8]. A major limitation of existing research on HAP and TB is the method of ascertainment of exposure and insufficient adjustment of confounders in analytical models. HAP is historically challenging to measure, and failure to use objective measures likely leads to misclassification and biased effect estimates[9]. Combustion produces a complex mixture of particulate, gaseous, and chemical constituents, of which the concentration of particulate matter less than 2.5 microns in diameter ($PM_{2.5}$) is most

commonly monitored and associated with negative health effects[8, 10, 11]. None of the existing research used $PM_{2.5}$ (or any other objective marker of combustion or differentiated $PM_{2.5}$) as the primary metric of exposure, relying solely on self-report of fuel use as the exposure definition[7, 8, 12]. Kerosene exposure has also not been included in most studies, which may also increase risk for TB through production of fine particulate products and chemical constituents during combustion[13-15].

India, like many high-burden TB countries, suffers a dual burden of TB and exposure to HAP. Along with shouldering the highest incident number of TB cases in the world and 27% of the total pediatric burden among the high burden countries, approximately 74% of households report using biomass fuels for cooking[2]. Further, India is estimated to have over 100 million smokers, the second largest number of smokers in the world[16]. Women of reproductive age, who are largely responsible for household cooking, and children, with developing lungs and immune systems, are especially vulnerable to HAP, often their largest source of air pollution exposure. Assessing the TB risk associated with exposure to HAP will provide needed evidence for intervention strategies among adults and children in India and other settings with these exposures. The purpose of this study is to estimate the association between HAP exposure and TB disease in adult women and children in India using strong, objective household concentrations of air pollution to more accurately classify participants' exposure in the home.

Methods

Ethics approval for this study was granted from the Institutional Review Boards of the Sassoon General Hospital and Byramjee Jeejeebhoy Medical College SGH/BJMC in Pune, Maharashtra, India and the Johns Hopkins University School of Medicine in Baltimore, Maryland, USA. All

participants, or primary caregivers of participants for children, provided written informed consent prior to participation in this study.

Study Population

Pune is a large district located in Maharasthra, a state in the western region of India. The district has a population of nearly 9.5 million, living in both urban (5.7 million) and rural (3.7 million) areas[17]. Research was conducted in partnership with SGH/BJMC Pune, India. SGH/BJMC is a large Maharashtra Government tertiary care public and teaching hospital, primarily serving the lower socioeconomic communities in Pune and surrounding peri-urban and rural areas. A matched case-control study was conducted to compare the odds of exposure to HAP among adult women with pulmonary TB and children under 5 years of age with TB as compared to geographically age and sex matched healthy controls.

Case Selection

Adult (≥ 18 years of age) women were eligible for inclusion in the study as index cases if they presented at the SGH/BJMC TB Control Program clinic and had two culture positive results or were GeneXpert positive. Women found to be HIV infected or who have diabetes were excluded. Children were included as cases if they presented at the SGH/BJMC TB Control Program clinic and had a diagnosis of confirmed, probable, or possible TB, using standard research definitions[18].

Control Selection

For adults, controls are healthy adult (≥ 18 years of age) women that were recruited at random from the same neighborhood and are within five years of age as the identified index case. For children, controls are healthy children within 12 months of age and of the same sex as the index case. Potential controls self-reporting previous diagnosis of HIV infection or diabetes were

excluded. Controls were also excluded if they tested positive for TB by symptom screen, which was defined as experiencing current fever, night sweats, weight loss, or prevalent cough for a period of 2 weeks, or (for children only) reporting any current fever, night sweats, weight loss, or prevalent cough if they had a reported exposure to a known case of TB within the previous 1 year[19]. For control selection, a random numbers table indicating direction on the street (left or right) and count of houses away from the index house was used to select potential control house. The counting for selected control homes began after 5 homes away from index home. If the household was unable to be enrolled, a note was made and the next available household on the street was approached. Next subsequent households were approached until a control was successfully enrolled.

All case and control participants, both adults and children, were excluded if they were not full-time residents of the home or had not lived in the home for the previous six months, or for the duration of their life if under six months of age.

Exposure Assessment

Exposure to air pollution from cooking fuels and SHS was measured in all cases and controls using structured questionnaires and objective measures of particulate matter less than 2.5 microns in diameter ($PM_{2.5}$), carbon monoxide (CO), and air nicotine.

All questionnaires were translated into Marathi in order to ensure that subjective questions would be asked in a standardized way, with response options that were clear to the participants, as well as to ensure that questions intended for validation exercises that were taken from existing published sources were true to their intention. Questionnaires were first translated into Marathi and back-translated into English. Discrepancies were identified, and further edits were made by

study team consensus. Minor changes were made to accommodate cultural understanding and local situations. Questionnaires assessing patterns of exposure both in the home and outside the home were administered to each participant, or their primary caregiver for the pediatric participants, at both the baseline and the 24-hour follow-up visit. At the baseline visit, participants were asked about typical use and exposure over the past 7 days regarding the types of fuel used in the home, whether the fuel was used to heat or cook, and the duration of use of these fuels. Information on ventilation was collected by asking participants whether they opened doors or windows when cooking. Additional reported exposures collected included trash burning near the home, exposure to neighbors using wood for cooking or regular preparation of Mishri (a smokeless tobacco product prepared by burning). Participants also reported the use of mosquito coils, incense, and candles or kerosene for lighting. Details of the housing construction and ventilation were recorded by observation. This included information on household characteristics such as construction materials of the walls, roof, and floor, the presence of a gap between the ceiling and the roof, the presence of a separate kitchen, and the size of the cooking space.

Details of household air monitoring procedures have been previously described (Dissertation Chapter 3). Briefly, household $PM_{2.5}$ was measured using the Thermo Environmental Instruments pDR-1000 sampler (Thermo Fisher Scientific, Waltham, MA) fitted with a cyclone inlet and paired with portable constant-flow pumps (SKC Inc, PA) and a downstream filter for $PM_{2.5}$ for gravimetric measurements. Lascar direct-reading CO monitors (EL-CO-USB 300, Lascar Electronics, Erie, PA) were also paired with the monitoring set-up, which was placed approximately 1 x 1 meter away from the primary cook stove in each home. Nephelometric measurements of $PM_{2.5}$ were collected every minute for a period of 24 hours, which were calibrated using gravimetric analysis of downstream filters.

Passive samplers for vapor-phase nicotine were used for the quantification of secondhand smoke in the home. The research team placed one air nicotine monitor in the home, which were left for a period of seven days, after which they were collected by research staff and stored in a smoke-free place until time of analysis. For quality control purposes, a 10% sample of blanks and a 10% sample of duplicates were included. Samples were analyzed at the JHSPH Secondhand Smoke Exposure Assessment Laboratory in Baltimore, MD, USA.

Statistical Analysis

The primary outcome of interest in this analysis is TB disease. Primary exposures of interest are 24-hour average household $PM_{2.5}$ and number of hours of $PM_{2.5}$ concentration > 75 ug/m^3, independently categorized into dichotomous variables for high and low exposure using the medians of each as the defined cut-off. This threshold was selected as it is an interim target for 24-hour $PM_{2.5}$ established by the World Health Organization (WHO)[11]. Socioeconomic information was collected using a structured questionnaire, which was then used to construct SES scores through principal component analysis (PCA) reduction with promax rotation (Supplemental Tables 2.S.1 – 2.S.6). Each household was assigned a score based on the PCA analysis results to control for SES. Descriptive statistical analysis was conducted to compared demographic and risk factor characteristics across case and control individuals as well as case and control households. Variables were compared across these groups using McNemar's (χ^2), Kruskal-Wallis, and Wilcoxon Rank Sum tests, as appropriate. Univariate odds ratios were first calculated, and covariates were considered for inclusion in the adjusted model if they were found to be significant in univariate analysis with $p<0.10$, or were considered to be of epidemiologic importance *a priori*. Single-pollutant, multivariate conditional logistic regression models were built for each of the primary exposures of interest with step-wise inclusion of eligible confounding variables to investigate the independent contribution of the primary risk factors of

interest to active TB disease. Further, exposure data were included in regression analysis as continuous variables to assess dose-response relationships. All multivariate models control for socioeconomic status using two principal component scores from PCA analysis (dichotomized into high and low using the median value), having a separate cooking area, always opening the doors when cooking, always keeping a window open when cooking, and crowding. When conducting analysis of the association of the primary cooking fuel with TB, we omitted households reporting wood as their primary source as they were few (n=4) and we did not feel it was appropriate to group them with kerosene in analyses (Tables 2.7 and 2.8 present regression results of the restricted data set).

Results

A total of 118 participants were recruited and enrolled into the study, including 32 pediatric cases less than 5 years of age and their matched controls, and 27 adult index cases and their matched controls. Among child participants, 40 (63%) were male and nearly all (n=58, 91%) reported BCG vaccination at birth. Participants primarily resided in low-income urban communities served by SGH/BJMC, and demographic and sociodemographic descriptive characteristics are presented in Table 2.1. The majority of participants lived in households with monthly incomes of less than 15,000 Indian Rupees (n=78, 22%), which is approximately $220 US Dollars, and 16 (14%) reported any food insecurity within the last 30 days. The highest proportion of participants lived in single-room households (n=51, 43%), with a median of 3 (IQR: 2 - 4) people per room, which we have defined as the threshold for crowding in this sample. Half (n=60, 51%) of the included homes were constructed with rudimentary materials (plastic, metal sheeting, or other rudimentary materials, as compared to brick, concrete, or wood), and 29 (25%) of household walls were made of rudimentary materials.

LPG was most often reported as the primary fuel source for the home (n=99, 84%) as compared to kerosene (n=15, 15%) and wood (n=4, 3%) (Table 2.2). Importantly, 45% of participants reported a secondary fuel source in the home, with the majority using kerosene (n=16, 14%) and wood (n=30, 25%) as supplemental fuels. In all, only 58 participants (n=49%) reported only using LPG in the home, which along with electricity is considered the only "clean" fuel source for this study. Wood was reported in 34 (29%) of homes, and kerosene (in the absence of wood) was reported in 26 (22%) of homes. None of the included participants reported using animal dung or crop waste as either a primary or a secondary fuel source. Nearly half of households reported cooking spaces that were not separated from other living spaces in the home (n=55, 47%).

Reported exposures to additional sources of HAP can be found in Table 2.4. Neighborhood burning of trash, either by the participants themselves or by their immediate neighbors, occurs at least weekly in 27 (22%) of households. Use of incense and mosquito coils were also commonly reported as used (n=105 (85%) and n=31 (25%), respectively). Kerosene as a lighting fuel source is used by 14 (11%) of included households, and over two thirds of homes reported using candles for lighting (n=84, 68%). No households reported using any fuel to heat their homes during cold months.

The median 24-hour time-weighted average $PM_{2.5}$ among all households was 184 (IQR: 107, 345), and was similar across case and control homes (Table 2.5). Average $PM_{2.5}$ categorized into quartiles and deciles were also similar across the two groups. Both groups also had high number of hours of exposure above 75 $\mu g/m^3$, with median levels reaching above 10 hours for both the case and the control groups. For each measure of time above this threshold, cases tended to have higher levels of exposure, however in a simple comparison this difference was not statistically significant.

Univariate and multivariate odds ratios for association between reported measures of exposure to cooking fuel and other household air pollutants with TB are shown in Tables 2.6-2.8. The use of kerosene as a primary cooking fuel source had a strong positive association with TB in both univariate (OR: 8.0; 95% CI: 1.0 - 64.0) and multivariate (OR 11.2, 95% CI: 1.2 - 100.8) analysis, with the additional sources of reported HAP included in the multivariate model. The composite variable for LPG only, kerosene use without any wood, and any wood use tended to show a positive association between kerosene and TB in adjusted analysis in both the full and the restricted data set (OR 3.2; 95% 0.8 - 13.3 and OR 3.4; 95% CI: 0.8 - 15.1, respectively).

In the restricted model, use of mosquito coils tended to have a positive association with TB, however this was not seen in the model using the full data set. In the full data set, the use of incense tended to be positively associated with TB, however the opposite trend was seen in the restricted data set.

Results of univariate and multivariate analysis of the association of pollutant concentrations with TB for all participants are presented in Table 2.9. No statistically significant difference was seen for log-unit increases in 24-average $PM_{2.5}$, although observations with averages above the median value tended to be positively associated with TB. In hourly threshold analysis, when dichotomized as above or below the median value of hours above 75 $\mu g/m^3$, a positive association was found (OR 3.30; 95% CI: 0.95 - 11.51) to approach statistical significance (p=0.06). Further, this association tended to hold with quartile increases in exposure (OR 1.72; 95% CI: 0.94 - 3.15; p=0.08). Levels of CO and detectable air nicotine were also evaluated in multivariate models, however were not associated with TB and did not alter the effect size of the primary pollutants of interest in the model (data not shown).

Sensitivity analysis stratified the participants by child and adult in independent regression models. While confidence intervals increased in these analyses, number of hours above the 75 $\mu g/m^3$ threshold continued to show signals of a positive association with TB (Table 2.10). Substantial increases in confidence intervals were seen in the results of multivariate analysis of adults alone, indicating perhaps greater variability in exposure among the sample of women. Effect sizes, however, continued to show strong positive associations between HAP and TB, approaching significance for several of the measures.

Discussion

This study assessed the association between levels of $PM_{2.5}$ and TB in homes of women and children with TB and their matched controls. To our knowledge this is the first study assessing the association between HAP and TB using objective markers of exposure to cooking fuel, as compared to subjective reported measures alone. Several measures of $PM_{2.5}$ were statistically significantly associated with TB in univariate analysis, and multivariate analysis approached statistical significance, suggesting that those with TB tended to have higher measures of HAP exposure as compared to healthy controls. Across all study homes, all participants were exposed to extremely high levels of $PM_{2.5}$, regardless of their reported fuel use (Dissertation Chapter 3).

The magnitude of the estimate of effect size found in the present study is higher than that found in systematic reviews for adults, however is consistent with many of the more recent studies published on the association using reported measures of exposure. Two recent systematic reviews have been published assessing the association between household air pollution and TB. Lin et al. showed a pooled odds ratio of 1.17 for case-control studies of solid fuel smoke and TB. The majority of the included studies, however, used weak statistical methods, leading to inconclusive

results[7]. One of the stronger studies was a case-control study of adult women and cooking fuel use in northern India. Here, Lakshmi et al. report an unadjusted OR of biomass versus LPG of 2.33, and an adjusted OR of 3.14 . This is similar to the effect estimate for our binary exposure category for number of hours above 75 μg/m^3 of $PM_{2.5}$[20]. Kerosene, however, was not included in the Lakshmi et al. study. In Nepal, kerosene used for cooking was found to be significantly associated with TB, with a three-fold increase in odds of TB. Kerosene as a lighting fuel source was much more strongly associated with TB, with the effect estimate three times higher than that for cooking, likely due to the longer use of the fuel during lighting as compared to cooking activities. The use of biomass fuel for cooking was not statistically significantly associated with, however when used as a heating fuel source was positively associated with TB (OR 3.45)[13]. Together, these results indicate that pollutant exposure from these sources, when found in high enough concentration, may put individuals at a higher risk for TB. Our results support this conclusion. In multivariate analysis comparing those reporting kerosene as their primary fuel source as compared to those with LPG as their primary fuel source, we also found a significant odds ratio of exposure. The effect of kerosene when included as a primary or a secondary fuel source tended to be associated with increased odds of TB, although no longer statistically significant perhaps due to the heterogeneity of exposure from combining primary and secondary usage.

Very few studies have been published assessing the association of HAP from cooking fuel sources with TB in children. A study amongst a similar population in Pune, India, found an increased odds of TB after adjustment using reported HAP exposure, however the definition of exposure was a composite variable including exposure to biomass fuels or reported SHS exposure in the home. Exposure to cooking fuel was defined by primary cooking fuel only, and no information on secondary fuel sources were collected. Our present study shows a high prevalence

of secondary fuels that may substantially contribute to household exposures. In separate analysis, biomass fuel use and SHS exposure alone tended to increase odds of TB, however this effect was not statistically significant[21]. A second case-control study among Indian children 0-14 years of age in Kerala found a seven-fold increase in odds for TB with exposure to wood smoke in multivariate analysis. Again, reported measures were used to assess exposure, and it is unclear what confounding variables were used in the adjusted model[22].

Our study did not find an association between household exposure to SHS and TB in contrast to prior studies. We defined exposure to SHS as detectable levels of nicotine from passive monitors placed in the home, as we have previously shown reported measures are not a valid measure of exposure in this population for the purpose of establishing an exposure-disease relationship (Dissertation Chapter 4). Pooled estimates from a recent meta-analysis, show an increase in risk for TB with exposure for both children and adults, and it may be that our sample size was too small to see an effect[23, 24]. Nearly all of the included studies, however, relied on reported measures of exposure to SHS, which may result in misclassification of exposure and biased results. Additional studies using environmental and/or biological markers of exposure would help strengthen the current evidence[8, 25]. The presence of SHS contributes to measures of $PM_{2.5}$, however, which we report to increase odds of TB disease.

The main strength of this study over previously published studies is the use of objective markers of air pollution exposure in the home, as compared to relying on self-reported exposure alone. Any exposure assessment runs the risk of misclassifying subjects, however we believe that understanding air pollution at the household level is an appropriate proxy for exposure both due to the importance of household exposure for women and children as well as the importance of the household as an intervention point for exposure reduction. Further, not only were we able to

capture objective 24-hour concentrations of exposure, but our direct-reading equipment allowed us to investigate the potential association with peak levels of exposure, resulting in evidence that households with a greater number of hours above the WHO interim target of 75 $\mu g/m^3$ may be at increased risk for TB. This analysis has results consistent with similar models evaluating peak levels of exposure (hours greater than 100 $\mu g/m^3$, a level chosen based on limit of detection of the monitoring equipment) with ALRI in children in Bangladesh[26].

Our study included adults as well as children, an often neglected and under-researched population in TB control. To account for the likely heterogenous effects of HAP for adult women and children, we conducted a stratified analysis, with adult and pediatric participants in separate models. In these stratified analyses, the tendency for a positive association between our measures of HAP and TB remained. While our adjusted effect sizes did not reach statistical significance, it is expected that with a larger sample size a statistically significant association would be found. The levels of air pollution found in homes using only LPG as a fuel source were surprisingly higher than anticipated, and likely muted the statistical effect we anticipated from fuels such as kerosene and wood. Classifying participants as "exposed" and "unexposed" is difficult in this setting with ubiquitously high exposures, and stronger measures of association would be anticipated in a setting where those categorized as having a "clean" fuel source have levels of exposure in the ranges we would have anticipated from use of LPG or electricity alone.

While we did not control for ambient air pollution in the current study with an independent variable in our regression model, the control population was matched to the cases on the neighborhood level. Thus, it is likely that ambient air pollution from neighboring roads, neighborhood combustion activity, and other sources such as coal-burning for electricity were controlled for in the matching analysis. Further, matched case and control participant households

were assessed for exposure during the same season, also accounting for changes in seasonality of exposures.

Like many other studies of TB risk factors, we are limited in the interpretation of study results due to our inability to control for malnutrition or to accurately characterize exposure to an active TB case. Due to extreme stigma around TB and issues of cultural sensitivity in this setting, testing for TB infection in community controls was not feasible. Collection of biological samples in this community is extremely difficult and often complicated by familiar pressure, as evidenced by the difficulty we faced in collecting hair and urine samples as part of this same protocol. Collection of blood or placement of TST was not feasible due to both logistical as well as financial reasons.

The results of this study add to the mounting evidence that HAP is a risk factor for TB disease in adults and children. Adult women and children are highly vulnerable to the negative health effects of HAP due to both the prevalence and intensity of exposure in the home. Children, with developing lungs, higher respiratory rates, and fragile immune systems, are particularly vulnerable. Additional research is needed to better quantify the true effect size of association with more statistical power. Additionally, understanding the exposure-response relationship is important for informing intervention strategies and the incremental benefit of lowered disease with reductions in HAP.

Table 2.1. Demographic and socioeconomic status characteristics of households of adult women and child index TB case participants and their matched controls (n=118 individuals, n=59 pairs) in Pune, India.

	Total (n=118)	**Control (n=59)**	**Case (n=59)**	**p-value†***	**Univariate OR (95% CI)**	**p-value***
Family Type, n(%)						
Nuclear	56 (47)	25 (42)	31 (53)	0.80	REF	
Joint/Extended	62 (53)	34 (58)	28 (47)		0.65 (0.30, 1.38)	0.26
Head of household is male, n(%)	46 (39)	26 (44)	20 (34)	0.14	0.63 (0.28, 1.38)	0.24
Years of education of head of household, n(%)						
< 4 years (primary)	72 (28)	**16 (26)**	**19 (31)**	**0.002**	REF	
≥ 4 years	46 (72)	**46 (74)**	**43 (69)**		0.67 (0.3, 1.48)	0.32
Employment status of head of household, n(%)						
Skilled/trained worker, housewife, or retired	71 (60)	39 (66)	32 (54)		REF	
Unskilled manual worker or unemployed	47 (40)	20 (20)	27 (27)	0.13	2.00 (0.81, 4.96)	0.13
Monthly household income, n(%)						
≤ 15,000 INR	95 (81)	**43 (73)**	**52 (88)**	**<0.0001**	REF	
> 15,000 INR	23 (19)	**16 (27)**	**7 (12)**		**0.36 (0.13, 0.99)**	**0.05**
Religion, n(%)						
Hindu	97 (82)	**49 (83)**	**48 (81)**	**< 0.0001**	REF	
Other	21 (18)	**10 (17)**	**11 (19)**		1.17 (0.39, 3.47)	0.78
Living location, n(%)						
Urban (non-slum)	26 (22)	**12 (20)**	**14 (24)**	**< 0.0001**	REF	
Urban slum, peri-urban, or rural	92 (78)	**47 (80)**	**45 (76)**		0.71 (0.23, 2.25)	0.57
Reported food insecurity[¥], n(%)	16 (14)	**6 (10)**	**10 (17)**	**< 0.0001**	1.80 (0.60, 5.37)	0.29
Crowding[π], n(%)	56 (47)	27 (46)	29 (49)	0.79	1.17 (0.54, 2.52)	0.70
Number of rooms (excluding bathroom), median (IQR)	2 (1, 2.0)	2 (1, 2)	2 (1, 2)	0.16	0.72 (0.47, 1.11)	0.13
Asset ownership, n(%)						
Clock or watch	104 (88)	53 (90)	51 (86)	**< 0.0001**	0.67 (0.19, 2.36)	0.53
Radio/CD player	50 (42)	24 (41)	26 (44)	0.29	1.15 (0.55, 2.42)	0.71
Television	96 (81)	46 (78)	50 (85)	**< 0.0001**	2.00 (0.60, 6.64)	0.26
Bicycle	34 (29)	14 (24)	20 (34)	**< 0.0001**	1.86 (0.74, 4.65)	0.19
Mobile phone	116 (98)	59 (100)	57 (97)	**< 0.0001**	--	--
Mattress	105 (89)	51 (86)	54 (92)	**< 0.0001**	1.75 (0.51, 5.98)	0.37
Chair	62 (53)	34 (58)	28 (47)	0.80	0.57 (0.24, 1.36)	0.21
Cot/bed	79 (67)	40 (68)	39 (66)	**0.01**	0.92 (0.40, 2.08)	0.83

Table	39 (33)	23 (39)	16 (27)	**0.02**	0.61 (0.29, 1.29)	0.2
Refrigerator	43 (36)	22 (37)	21 (36)	0.052	0.92 (0.42, 2.02)	0.84
Motorcycle/scooter	51 (43)	30 (51)	21 (36)	0.40	0.50 (0.23, 1.11)	0.09
Electric fan	114 (97)	59 (100)	55 (93)	**< 0.0001**	--	--
Car	6 (5)	5 (8)	1 (2)	**< 0.0001**	0.20 (0.02, 1.71)	0.14
Washing machine	4 (3)	2 (3)	2 (3)	**< 0.0001**	--	--
Pressure cooker	110 (93)	55 (93)	55 (93)	**< 0.0001**	--	--
Sewing machine	24 (20)	14 (24)	10 (17)	**< 0.0001**	0.64 (0.25, 1.64)	0.35
Asset Index (number of assets owned), median (IQR)	9 (7, 10)	9 (7, 11)	9 (7, 10)	0.34	0.90 (0.75, 1.08)	0.27
Ownership of additional land, n(%)	43 (36)	**21 (36)**	**22 (37)**	**0.05**	1.09 (0.48, 2.47)	0.83
Ownership status of home, n(%)						
Owned	60 (51)	32 (54)	28 (47)	1.00	REF	
Not owned	58 (49)	27 (46)	31 (53)		1.50 (0.61, 3.67)	0.37
Roofing material plastic, rudimentary, or metal sheet[f], n(%)	60 (51)	28 (47)	32 (54)	1.00	1.80 (0.60, 5.37)	0.29
Exterior walls made of bamboo/mud or metal sheets[f], n(%)	29 (25)	**13 (22)**	**16 (27)**	**< 0.001**	2.00 (0.50, 8.00	0.33
Flooring concrete or rudimentary (no tiles), n(%)	45 (38)	22 (37)	23 (39)	**0.09**	1.14 (0.41, 3.15)	0.80
Presence of dampness and/or condensation, n(%)	58 (50)	32 (54)	26 (45)	1	0.58 (0.23, 1.48)	0.26
Electricity supply shared/illegal/none, n(%)	33 (28)	**12 (20)**	**21 (36)**	**< 0.001**	**2.80 (1.01, 7.77)**	**0.05**
Own toilet facility, n(%)	55 (47)	30 (51)	25 (42)	0.71	0.55 (0.20, 1.47)	0.23
Housing condition, n(%)						
Sound structure	84 (71)	**44 (75)**	**40 (68)**	**0.001**	REF	
Need of repairs	34 (29)	**15 (25)**	**19 (32)**		1.67 (0.61, 4.59)	0.32
Health insurance, n(%)	23 (19)	17 (29)	6 (10)	**< 0.0001**	**0.15 (0.03, 0.68)**	**0.01**
Life Insurance, n(%)	43 (36)	25 (42)	18 (31)	0.06	0.50 (0.20, 1.24)	0.13
Bank account, n(%)	100 (85)	52 (88)	48 (81)	**< 0.0001**	0.56 (0.19, 1.66)	0.29

†McNemar test, Kruskal-Wallis test, Wilcox Rank Sum test

*****Bolded values are statistically significant at $p < 0.05$

¥ As measured by the Household Food Insecurity Assessment Survey (HFIAS). Scores ≥ 1 categorized as insecure.

π Greater than the median number of people per room in this sample (median = 3.25 people/room)

f Compared to brick, concrete, or wood

Table 2.2. Descriptive statistics about cooking fuel use and cooking-related exposures in households of adult women and child index TB case participants and their matched controls (n=118 individuals, n=59 pairs) in Pune, India.

	Total (n=118)	**Control (n=59)**	**Case (n=59)**	**p-value†***	**Univariate OR (95% CI)**	**p-value***
Primary fuel types, n(%)						
LPG or electricity	99 (84)	52 (88)	47 (80)		REF	
Kerosene	15 (13)	3 (5)	12 (20)	0.30	8.00 (1.0, 64)	0.05
Wood	4 (3)	4 (7)	0 (--)		--	--
Secondary fuel types, n(%)						
None	65 (55)	35 (59)	30 (51)			
LPG or electricity	7 (6)	3 (5)	4 (7)		REF	
Kerosene	16 (14)	7 (12)	9 (15)	0.42	1.65 (0.45, 6.06)	0.45
Wood	30 (25)	14 (24)	16 (27)		1.36 (0.52, 3.55)	0.53
Composite Fuel Types¥, n(%)						
LPG/electricity only	58 (49)	32 (54)	26 (44)		REF	
Kerosene (no biomass)	26 (22)	9 (15)	17 (29)	0.57	2.76 (0.91, 8.42)	0.07
Any biomass	34 (29)	18 (31)	16 (27)		1.17 (0.42, 3.28)	0.77
Number of hours of cooking per day, median (IQR)	2.3 (1.5, 3.3)	2.5 (1.8, 3.3)	2.0 (1.5, 3.1)	0.32	0.84 (0.61, 1.16)	0.28
Average use of primary cooking fuel, n(%)						
Less than 30 minutes	1 (1)	0 (--)	1 (2)			
30 minutes to 1 hour	2 (2)	1 (2)	1 (2)			
Between 1-2 hours	9 (8)	6 (10)	3 (5)	0.06	0.71 (0.46, 1.09)	0.11
More than 2 to 3 hours	46 (39)	16 (27)	30 (51)			
More than 3 to 5 hours	50 (42)	29 (49)	21 (36)			
More than 5 hours	10 (8)	7 (12)	3 (5)			
Use of primary cook fuel more than 3 hours per day, n(%)	60 (51)	36 (61)	24 (41)	1.0	**0.46 (0.22, 0.96)**	**0.04**
Average use of secondary cook fuel per day^{ß}, n(%)						
Less than 30 minutes	5 (6)	2 (5)	3 (7)			
30 minutes to 1 hour	16 (18)	8 (20)	8 (17)			
Between 1-2 hours	10 (11)	6 (15)	4 (9)	0.99	1.02 (0.80, 1.30)	0.85
More than 2 to 3 hours	11 (13)	5 (12)	6 (13)			
More than 3 to 5 hours	7 (8)	1 (2)	6 (13)			
Always open the windows when cooking, n(%)	55 (47)	28 (47)	27 (47)	0.70	0.93 (0.44, 1.98)	0.85
Always open the doors when cooking, n(%)	81 (69)	**43 (73)**	**38 (64)**	**0.008**	0.62 (0.26, 1.48)	0.28
No separate cooking area from living space, n(%)	55 (47)	27 (53)	28 (48)	0.70	1.08 (0.49, 2.37)	0.84

Cooking area volume, n(%)	405 (28, 741)	399 (36, 776)	405 (28, 702)	0.59	--	--
Child near when cooking (among children), n(%)						
Never	37 (58)	19 (59)	18 (56)		REF	
At least sometimes	27 (42)	13 (41)	14 (44)	0.47	1.20 (0.37, 3.93)	0.76
Responsible for all or most of cooking (among adults), n(%)						
No	17 (31)	1 (4)	16 (59)		REF	
Yes	37 (69)	26 (96)	11 (41)	0.16	**0.06 (0.01, 0.47)**	**0.007**

†McNemar test, Kruskal-Wallis test, Wilcox Rank Sum test

*Bolded values are statistically significant at $p < 0.05$

¥ Combined variable indicating both primary and secondary fuel source.

ß Among those reporting a secondary cooking fuel

Table 2.3. Descriptive statistics of ventilation and cooking area characteristics in households of adult women and child index TB case participants and their matched controls (n=118 individuals, n=59 pairs) in Pune, India.

	Total (n=118)	**Control (n=59)**	**Case (n=59)**
Cooking area, n(%)			
Outside	2 (2)	1 (2)	1 (2)
Inside, separate kitchen	61 (52)	31 (52)	30 (51)
Inside, no separate kitchen	55 (47)	27 (46)	28 (47)
Cooking Area, binary, n(%)			
Outside or separate kitchen	63 (53)	32 (54)	31 (53)
Inside, no separate kitchen	55 (47)	27 (46)	28 (47)
Type of fuel being monitored, n(%)			
LPG	98 (83)	53 (90)	45 (76)
Paraffin/kerosene	15 (13)	3 (5)	12 (20)
Coal	0 (--)	0 (--)	0 (--)
Electricity	1 (1)	0 (--)	1 (2)
Wood	4 (3)	3 (5)	1 23)
Animal Dung	0 (--)	0 (--)	0 (--)
Crop Waste	0 (--)	0 (--)	0 (--)
Primary construction material of the kitchen walls, n(%)			
Not applicable, outdoor kitchen	0 (--)	0 (--)	0 (--)
Concrete	87 (74)	45 (76)	42 (71)
Brick	6 (5)	2 (3)	4 (7)
Corrugated Metal	25 (21)	12 (20)	13 (22)
Wood	0 (--)	0 (--)	0 (--)
Thatch	0 (--)	0 (--)	0 (--)
Primary construction material of the kitchen roof, n(%)			
Not applicable, outdoor kitchen	1 (1)	1 (2)	0 (--)
Concrete	57 (48)	29 (49)	28 (47)
Brick	0 (--)	0 (--)	0 (--)
Corrugated metal	51 (43)	25 (42)	26 (44)
Wood	9 (8)	4 (7)	5 (19)
Thatch	0 (--)	0 (--)	0 (--)
Doors opening to the outside areas are made of: n(%)			
No doors opening to the outside	7 (6)	4 (7)	3 (5)

Not applicable, hollow/always open	10 (9)	3 (5)	7 (12)
Cloth, bamboo, or other soft material	2 (2)	2 (3)	0 (--)
Wood, glass, or other hard material	98 (84)	49 (84)	49 (83)
Doors opening to the inside areas are made of: n(%)			
No doors opening to the inside	60 (51)	28 (48)	32 (54)
Not applicable, hollow/always open	45 (38)	23 (40)	22 (37)
Cloth, bamboo, or other soft material	0 (--)	0 (--)	0 (--)
Wood, glass, or other hard material	12 (11)	7 (12)	5 (9)
Windows opening to the outside areas are made of: n(%)			
No windows opening to the outside	50 (43)	25 (43)	25 (42)
Hollow/always open	22 (19)	12 (21)	10 (17)
Cloth, bamboo, or other soft material	2 (2)	1 (2)	1 (2)
Wood, glass, or other hard material	43 (37)	20 (34)	23 (39)
Visible gap between the roof and the top of the walls, n(%)	23 (20)	9 (16)	14 (24)
Size of gap among those with gap (cm), median (IQR)	4 (3, 6)	5 (4, 9)	4 (3, 5)

†McNemar test, Kruskal-Wallis test, Wilcox Rank Sum test

*Bolded values are statistically significant at $p < 0.05$

Table 2.4. Reported exposure to additional sources of household air pollution in households of adult women and child index TB case participants and their matched controls (n=118 individuals, n=59 pairs) in Pune, India.

	Total (n=118)	**Control (n=59)**	**Case (n=59)**	**p-value†***	**Univariate OR (95% CI)**	**p-value***
Burning of trash near home, n(%)						
Less than weekly	97 (78)	**49 (79)**	**48 (77)**		REF	
At least weekly	27 (22)	**13 (21)**	**14 (23)**	**< 0.0001**	1.13 (0.43, 2.92)	0.81
Burning incense, n(%)						
No	19 (15)	**8 (13)**	**11 (18)**		REF	
Yes	105 (85)	**54 (87)**	**51 (82)**	**< 0.0001**	0.67 (0.24, 1.87)	0.44
Use mosquito coils, n(%)						
No	93 (75)	**49 (79)**	**44 (71)**		REF	
Yes	31 (25)	**13 (21)**	**18 (29)**	**< 0.0001**	1.63 (0.67, 3.92)	0.28
Kerosene light source, n(%)						
No	110 (89)	**55 (89)**	**55 (89)**		REF	
Yes	14 (11)	**7 (11)**	**7 (11)**	**< 0.0001**	1.00 (0.29, 3.45)	1.00
Candle light source, n(%)						
No	40 (32)	**22 (35)**	**18 (29)**			
Yes	84 (68)	**40 (65)**	**44 (71)**	**0.003**	1.44 (0.62, 3.38)	0.4

†McNemar test, Kruskal-Wallis test, Wilcox Rank Sum test
*Bolded values are statistically significant at $p < 0.05$

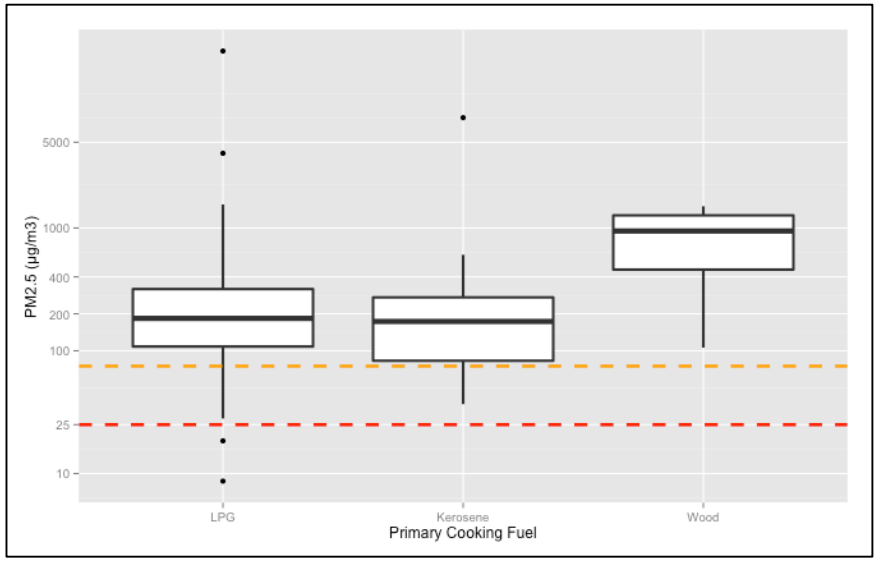
Table 2.5. Concentration of $PM_{2.5}$, CO, and air nicotine in households of adult women and child index TB case participants and their matched controls (n=118 individuals, n=59 pairs) in Pune, India.

	Total (n=118)	**Control (n=59)**	**Case (n=59)**	**p-value***
Mean 24-hour $PM_{2.5}$ ($\mu g/m^3$), median (IQR)	184 (107, 345)	182 (108, 305)	128 (107, 383)	0.60
Quartile of mean 24-hour $PM_{2.5}$ ($\mu g/m^3$), n(%)				
1 [8.67, 107]	30 (25)	15 (25)	15 (25)	0.57
2 (107, 184]	29 (25)	16 (27)	13 (22)	
3 (184, 344]	29 (25)	15 (25)	14 (23)	
4 (344, 27700]	30 (25)	13 (22)	17 (29)	
Decile of mean 24-hour $PM_{2.5}$ ($\mu g/m^3$), median (IQR)	5.5 (3.0, 8.0)	5.0 (3.0, 8.0)	6.0 (3.0, 8.0)	0.55
Log 24-hour $PM_{2.5}$ ($\mu g/m^3$), mean (SD)	5.3 (1.1)	5.2 (1.1)	5.4 (1.2)	0.60
Hours of $PM_{2.5}$ > 75 $\mu g/m^3$, median (IQR)	11.6 (5.4, 18.8)	10.5 (5.2, 18.5)	12.0 (5.9, 19.3)	0.44
Quartile of hours of $PM_{2.5}$ > 75 $\mu g/m^3$, n(%)				
1 [0.0667,4.27]	30 (25)	17 (29)	13 (22)	0.22
2 (4.27,9.74]	29 (25)	17 (29)	12 (20)	
3 (9.74,16.5]	29 (25)	11 (19)	18 (31)	
4 (16.5,24]	30 (25)	14 (24)	16 (27)	
Decile of hours of $PM_{2.5}$ > 75 $\mu g/m^3$, median (IQR)	5.5 (3.0, 8.0)	5.0 (3.0, 8.0)	6.0 (3.0, 8.0)	0.59
Tobacco-specific Measure (n=98 observations)¥				
Detectable air nicotine				
No	64 (65)	**31 (65)**	**33 (66)**	**0.03**
Yes	34 (35)	**17 (35)**	**17 (34)**	
Air nicotine ($\mu g/m^3$), median (IQR)	0.001 (0.001, 0.01	0.001 (0.001, 0.01)	0.001 (0.001, 0.02)	0.81

*Bolded values are statistically significant at $p < 0.05$

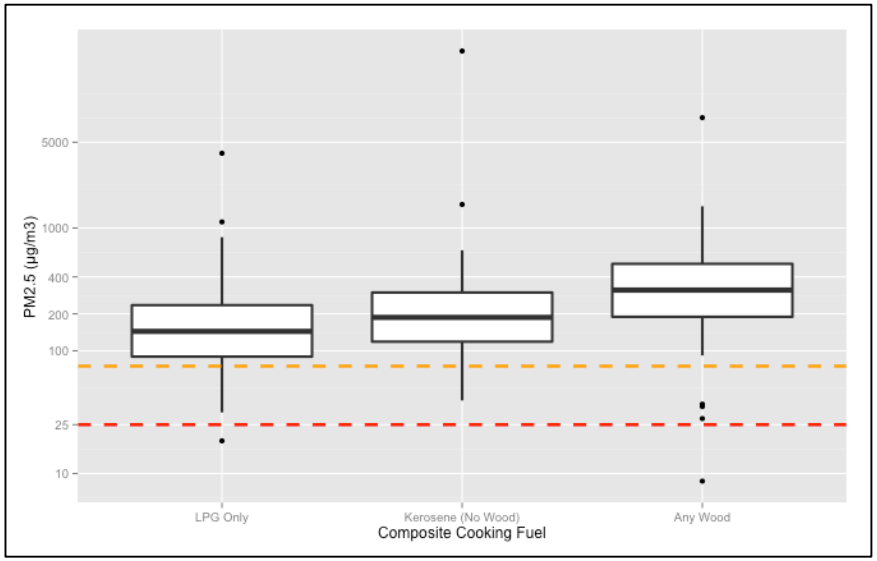
¥ Regression excluding observations without air nicotine exposure due to damage of mointor

Figure 2.1. Mean 24-hour average $PM_{2.5}$ concentration ($\mu g/m^3$) by type of primary cooking fuel in households of adult women and child index TB case participants and their matched controls (n=118 individuals, n=59 pairs) in Pune, India.



† Red and orange lines indicate WHO 24-hour average exposure and interim target levels, respectively, for $PM_{2.5}$.

Figure 2.2. Mean 24-hour average $PM_{2.5}$ concentration ($\mu g/m^3$) by composite cooking fuel in households of adult women and child index TB case participants and their matched controls (n=118 individuals, n=59 pairs) in Pune, India.



† Red and orange lines indicate WHO 24-hour average exposure and interim target levels, respectively, for $PM_{2.5}$.

Table 2.6. Univariate and multivariate conditional logistic regression for TB outcome and reported measures of exposure to composite cooking fuels and other sources of HAP as independent variables among adult women and child index TB case participants and their matched controls (n=118 individuals, n=59 pairs).

	Control (n=59)	**Case (n=59)**	**Univariate OR (95% CI)**	**p-value***	**Multivariate¥ OR (95% CI)**	**p-value***
Cooking fuel (composite)						
LPG or electricity only	32 (54)	26 (44)	REF		REF	
Kerosene (no wood)	9 (15)	17 (29)	2.76 (0.91, 8.42)	0.07	3.21 (0.78, 13.26)	0.11
Any wood	18 (31)	16 (27)	1.17 (0.42, 3.28)	0.77	1.02 (0.32, 3.22)	0.98
Burning of trash near home, n(%)						
Less than weekly	49 (79)	48 (77)	REF		REF	
At least weekly	13 (21)	14 (23)	1.13 (0.43, 2.92)	0.81	0.98 (0.26, 3.62)	0.97
Burning incense, n(%)						
No	8 (13)	11 (18)	REF		REF	
Yes	54 (87)	51 (82)	0.67 (0.24, 1.87)	0.44	2.71 (0.75, 9.83)	0.13
Use mosquito coils, n(%)						
No	49 (79)	44 (71)	REF		REF	
Yes	13 (21)	18 (29)	1.63 (0.67, 3.92)	0.28	0.50 (0.12, 2.09)	0.34
Kerosene light source, n(%)						
No	55 (89)	55 (89)	REF		REF	
Yes	7 (11)	7 (11)	1.00 (0.29, 3.45)	1.00	0.22 (0.02, 1.91)	0.17

* Bolded values statistically significant at $p < 0.05$

¥ Multivariate analysis also controlling for Principal Component 1 Score above or below the median value, Principal Component 3 Score above or below the median value, having a separate cooking space, always opening the door when cooking, always keeping a window open when cooking, and crowding

Table 2.7. Univariate and multivariate conditional logistic regression for TB outcome and reported measures of exposure to primary cooking fuels and other sources of HAP as independent variables among adult women and child index TB case participants and their matched controls, restricting to those not using wood as a primary fuel source (n=110 individuals, n=55 pairs).

	Control (n=55)	**Case (n=55)**	**Univariate OR (95% CI)**	**p-value***	**Multivariate¥ OR (95% CI)**	**p-value***
Primary cooking fuel						
LPG or electricity	52 (95)	45 (45)	REF		REF	
Kerosene	3 (5)	10 (10)	**8.00 (1.00, 63.96)**	**0.05**	**11.18 (1.24, 100.8)**	**0.04**
Burning of trash near home, n(%)						
Less than weekly	44 (80)	42 (76)	REF		REF	
At least weekly	11 (20)	13 (24)	1.33 (0.46, 3.84)	0.58	0.67 (0.16, 2.79)	0.59
Burning incense, n(%)						
No	6 (11)	10 (19)	REF		REF	
Yes	49 (89)	45 (82)	0.56 (0.19, 1.66)	0.29	0.36 (0.08, 1.60)	0.18
Use mosquito coils, n(%)						
No	43 (78)	40 (73)	REF		REF	
Yes	12 (22)	15 (27)	1.43 (0.54, 3.75)	0.47	3.20 (0.81, 12.62)	0.10
Kerosene light source, n(%)						
No	49 (89)	52 (95)	REF		REF	
Yes	6 (11)	3 (5)	0.40 (0.08, 2.06)	0.27	0.18 (0.02, 1.91)	0.15

* Bolded values statistically significant at $p < 0.05$

¥ Multivariate analysis also controlling for Principal Component 1 Score above or below the median value, Principal Component 3 Score above or below the median value, always opening the door when cooking, always keeping a window open when cooking, and crowding

Table 2.8. Univariate and multivariate conditional logistic regression for TB outcome and reported measures of exposure to composite cooking fuel and other sources of HAP as independent variables among adult women and child index TB case participants and their matched controls, restricting to those not using wood as a primary fuel source (n=110 individuals, n=55 pairs).

	Control (n=55)	**Case (n=55)**	**Univariate OR (95% CI)**	**p-value***	**Multivariate¥ OR (95% CI)**	**p-value***
Cooking Fuel (composite)						
LPG/electricity only	32 (58)	26 (47)	REF		REF	
Kerosene (no wood)	9 (16)	15 (27)	2.40 (0.79, 7.32)	0.12	3.43 (0.78, 15.09)	0.10
Any wood	14 (25)	14 (25)	1.31 (0.46, 3.75)	0.62	1.15 (0.36, 3.71)	0.81
Burning of trash near home, n(%)						
Less than weekly	44 (80)	42 (76)	REF		REF	
At least weekly	11 (20)	13 (24)	1.33 (0.46, 3.84)	0.58	1.02 (0.26, 4.03)	0.98
Burning incense, n(%)						
No	6 (11)	10 (19)	REF		REF	
Yes	49 (89)	45 (82)	0.56 (0.19, 1.66)	0.29	0.48 (0.12, 1.95)	0.30
Use mosquito coils, n(%)						
No	43 (78)	40 (73)	REF		REF	
Yes	12 (22)	15 (27)	1.43 (0.54, 3.75)	0.47	2.27 (0.60, 8.65)	0.23
Kerosene light source, n(%)						
No	49 (89)	52 (95)	REF		REF	
Yes	6 (11)	3 (5)	0.40 (0.08, 2.06)	0.27	0.13 (0.01, 1.65)	0.11

* Bolded values statistically significant at $p < 0.05$

¥ Multivariate analysis also controlling for Principal Component 1 Score above or below the median value, Principal Component 3 Score above or below the median value, always opening the door when cooking, always keeping a window open when cooking, and crowding

Table 2.9. Univariate and multivariate conditional logistic regression for TB outcome and household measures of HAP as primary exposures of interest among adult women and child index TB case participants and their matched controls (n=118 individuals, n=59 pairs) in independent models.

	Control (n=59)	**Case (n=59)**	**Univariate OR (95% CI)**	**p-value***	**Multivariate¥ OR (95% CI)**	**p-value***
Log 24-hour $PM_{2.5}$ (μg/m^3), mean (SD)						
≤ Mean (5.3 μg/m^3)	34 (58)	28 (47)	REF		REF	
> Mean	25 (42)	31 (53)	1.86 (0.74, 4.65)	0.19	2.01 (0.70, 5.81)	0.20
Log 24-hour average $PM_{2.5}$ (μg/m^3), mean (SD)	5.2 (1.1)	5.4 (1.2)	1.18 (0.80, 1.75)	0.40	1.22 (0.79, 1.89)	0.38
Quartile of log 24-hour average $PM_{2.5}$ (μg/m^3), n(%)						
1 [2.16,4.68]	15 (25)	15 (25)	REF		REF	
2 (4.68,5.21]	16 (27)	13 (22)	0.83 (0.30, 2.36)	0.73	0.76 (0.23, 2.48)	0.65
3 (5.21,5.84]	15 (25)	14 (24)	0.98 (0.31, 3.13)	0.97	0.81 (0.21, 3.08)	0.76
4 (5.84,10.2]	13 (22)	17 (29)	1.41 (0.44, 4.51)	0.56	1.44 (0.39, 5.33)	0.58
Log 24-hour average $PM_{2.5}$ (μg/m^3), continuous quartile, median (IQR)	2.0 (1.5, 3.0)	3.0 (1.5, 4.0)	1.4 (0.78, 1.65)	0.51	1.13 (0.74, 1.74)	0.56
Log 24-hour average $PM_{2.5}$ (μg/m^3) greater than mean among those with LPG as the primary cooking fuel						
≤ Mean (5.3)	33 (56)	28 (48)	REF		REF	
> Mean	26 (44)	31 (53)	1.63 (0.67, 3.92)	0.28	1.76 (0.64, 4.89)	0.28
Log 24-hour average $PM_{2.5}$ (μg/m^3) greater than mean among those with LPG as only cooking fuel						
≤ Mean (5.1)	26 (44)	26 (44)	REF		REF	
> Mean	33 (56)	33 (56)	1.00 (0.43, 2.31)	1.00	0.84 (0.32, 2.20)	0.72
Hours of $PM_{2.5}$ > 75 μg/m^3, n(%)						
≤ Median (11.6 hours)	34 (58)	25 (42)	REF		REF	
> Median	25 (42)	34 (58)	**3.25 (1.06, 9.97)**	**0.04**	3.30 (0.95, 11.51)	0.06
Hours of $PM_{2.5}$ > 75 μg/m^3, median (IQR)	10.5 (5.2, 18.5)	12.0 (5.9, 19.3)	1.04 (0.96, 1.13)	0.33	1.03 (0.95, 1.13)	0.46
Quartile of hours of $PM_{2.5}$ > 75 μg/m^3, n(%)						
1 [0.183,5.44]	17 (29)	13 (22)	REF		REF	
2 (5.44,11.6]	17 (29)	12 (20)	1.27 (0.40, 4.08)	0.69	1.30 (0.36, 4.64)	0.69
3 (11.6,18.8]	11 (19)	18 (31)	4.13 (0.95, 18.00)	0.06	3.85 (0.78, 18.95)	0.10

4 (18.8,24]	14 (24)	16 (27)	3.42 (0.72, 16.36)	0.12	4.29 (0.66, 27.86)	0.13
Quartile of hours of $PM_{2.5}$ > 75 μg/m^3 as continuous, median (IQR)	2.0 (1.0, 3.0)	3.0 (2.0, 4.0)	1.55 (0.94, 2.55)	0.08	1.72 (0.94, 3.15)	0.08
Hours of $PM_{2.5}$ > 75 μg/m3 among those with LPG as the primary cooking fuel, n(%)						
≤ Median (11.7 hours)	35 (59)	27 (46)	REF		REF	
> Median	24 (41)	32 (54)	3.00 (0.97, 9.3)	0.06	3.01 (0.88, 10.30)	0.08
Hours of $PM_{2.5}$ > 75 μg/m3 among those with LPG as the only cooking fuel, n(%)						
≤ Median (11.7 hours)	35 (59)	27 (46)	REF		REF	
> Median	24 (41)	32 (54)	3.00 (0.97, 9.3)	0.06	3.01 (0.88, 10.30)	0.08

***** Bolded values statistically significant at $p < 0.05$

¥ Multivariate analysis controlling for Principal Component 1 Score above or below the median value, Principal Component 3 Score above or below the median value, having a separate cooking space, always opening the door when cooking, always keeping a window open when cooking, and crowding

Table 2.10. Univariate and multivariate conditional logistic regression for TB outcome and household measures of HAP as primary exposures of interest among child index TB case participants and their matched controls (n=64 individuals, n=32 pairs) in independent models.

	Control n=32	**Case n=32**	**OR (95% CI)**	**p-value**	**Multivariate OR (95% CI)**	**p-value**
Log 24-hour average $PM_{2.5}$ ($\mu g/m^3$)						
≤ Mean (5.295197)	15 (47)	10 (31)	REF		REF	
> Mean	17 (53)	22 (69)	2.67 (0.71, 10.05)	0.15	3.19 (0.66, 15.46)	0.15
Log 24-hour average $PM_{2.5}$ ($\mu g/m^3$), mean (sd)	5.3 (1.1)	5.7 (1.1)	1.39 (0.83, 2.32)	0.22	1.41 (0.74, 2.69)	0.30
Log 24-hour average $PM_{2.5}$ ($\mu g/m^3$), quartile n(%)						
1 [2.16,4.68]	9 (25)	5 (16)	REF		REF	
2 (4.68,5.21]	7 (22)	5 (16)	1.42 (0.20, 9.97)	0.73	1.32 (0.02, 4.23)	0.38
3 (5.21,5.84]	7 (22)	8 (25)	3.10 (0.42, 22.8)	0.27	1.20 (0.13, 14.63)	0.78
4 (5.84,10.2]	10 (31)	14 (44)	3.31 (0.56, 19.62)	0.19	1.89 (0.24, 14.84)	0.54
Log 24-hour average $PM_{2.5}$ ($\mu g/m^3$), continuous quartile, median (IQR)	3.0 (1.8, 4.0)	3.0 (2.0, 4.0)	1.46 (0.86, 2.47)	0.16	1.47 (0.79, 2.76)	0.23
Log 24-hour average $PM_{2.5}$ ($\mu g/m^3$) greater than mean for LPG as primary cooking fuel						
≤ Mean (5.254183)	15 (47)	10 (31)	REF		REF	
> Mean	17 (53)	22 (69)	2.67 (0.71, 10.05)	0.15	3.19 (0.66, 15.46)	0.15
Log 24-hour average $PM_{2.5}$ ($\mu g/m^3$) greater than mean for LPG as only cooking fuel						
≤ Mean (5.140688)	13 (41)	9 (28)	REF		REF	
> Mean	19 (59)	23 (72)	2.00 (0.60, 6.64)	0.26	2.04 (0.50, 8.24)	0.32
Hours greater than 75 $\mu g/m^3$ $PM_{2.5}$						
≤ Median (11.575hours)	17 (53)	10 (31)	REF		REF	
> Median	15 (47)	22 (69)	4.50 (0.97, 20.83)	0.05	4.23 (0.83, 21.68)	0.08
Hours greater than 75 $\mu g/m^3$ $PM_{2.5}$, median (IQR)	11.4 (8.5, 18.7)	17.1 (9.7, 20.2	1.07 (0.96, 1.19)	0.20	1.06 (0.94, 1.20)	0.36
Hours greater than 75 $\mu g/m^3$ $PM_{2.5}$, quartile						
1 [0.183,5.44]	7 (22)	4 (13)	REF		REF	
2 (5.44,11.6]	10 (31)	6 (19)	1.29 (0.20, 8.37)	0.79	1.05 (0.11, 9.98)	0.96
3 (11.6,18.8]	7 (22)	9 (28)	4.29 (0.57, 32.16)	0.15	3.44 (0.37, 31.59)	0.28
4 (18.8,24]	8 (25)	13 (41)	7.10 (0.79, 63.81)	0.08	6.79 (0.53, 86.83)	0.14

Hours greater than 75 μg/m^3 $PM_{2.5}$, quartile (continuous)	2.0 (2.0, 3.3)	3.0 (2.0, 4.0)	**2.03 (1.01, 4.09)**	**0.05**	2.06 (0.93, 4.56)	0.08
Hours greater than 75 μg/m^3 $PM_{2.5}$ for LPG as primary cook fuel						
≤ Median (11.65 hours)	17 (53)	12 (38)	REF		REF	
> Median	15 (47)	20 (63)	2.67 (0.71, 10.05)	0.15	2.69 (0.64, 11.37)	0.18
Hours greater than 75 μg/m^3 $PM_{2.5}$ for LPG as only cooking fuel						
≤ Median (11.65 hours)	17 (53)	12 (38)	REF		REF	
> Median	15 (47)	20 (63)	2.67 (0.71, 10.05)	0.15	2.69 (0.64, 11.37)	0.18

***** Bolded values statistically significant at $p < 0.05$

¥ Multivariate analysis controlling for Principal Component 1 Score above or below the median value, Principal Component 3 Score above or below the median value, having a separate cooking space, always opening the door when cooking, always keeping a window open when cooking, and crowding

Table 2.11. Univariate and multivariate conditional logistic regression for TB outcome and household measures of HAP as primary exposures of interest among adult women index TB case participants and their matched controls (n=654individuals, n=27 pairs) in independent models.

	Control n=27	**Case n=27**	**OR (95% CI)**	**p-value**	**Multivariate OR (95% CI)**	**p-value**
Log 24-hour average $PM_{2.5}$ ($\mu g/m^3$)						
≤ Mean (5.295197)	19 (70)	18 (67)	REF		REF	
> Mean	8 (30)	9 (33)	1.25 (0.34, 4.66)	0.74	1.69 (0.25, 11.43)	0.59
Log 24-hour average $PM_{2.5}$ ($\mu g/m^3$), mean (sd)	5.1 (1.0)	5.0 (1.3)	0.85 (0.41, 1.73)	0.65	0.83 (0.34, 1.95)	0.68
Log 24-hour average $PM_{2.5}$ ($\mu g/m^3$), quartile n(%)						
1 [2.16,4.68]	7 (26)	10 (37)	REF		REF	
2 (4.68,5.21]	9 (33)	8 (30)	0.61 (0.17, 2.24)	0.46	0.60 (0.10, 3.58)	0.58
3 (5.21,5.84]	8 (30)	6 (22)	0.47 (0.10, 2.27)	0.35	0.64 (0.09, 4.79)	0.67
4 (5.84,10.2]	3 (11)	3 (11)	0.65 (007, 5.78)	0.70	0.29 (0.01, 6.23)	0.43
Log 24-hour average $PM_{2.5}$ ($\mu g/m^3$), continuous quartile, median (IQR)	2.0 (1.5, 3.0)	2.0 (1.0, 3.0)	0.79 (0.43, 1.46)	0.45	0.73 (0.33, 1.59)	0.42
Log 24-hour average $PM_{2.5}$ ($\mu g/m^3$) greater than mean for LPG as primary cooking fuel						
≤ Mean (5.254183)	18 (67)	18 (67)	REF		REF	
> Mean	9 (33)	9 (33)	1.00 (0.29, 3.45)	1.00	1.47 (0.23, 9.31)	0.68
Log 24-hour average $PM_{2.5}$ ($\mu g/m^3$) greater than mean for LPG as only cooking fuel						
≤ Mean (5.140688)	13 (48)	17 (63)	REF		REF	
> Mean	14 (52)	10 (37)	0.43 (0.11, 1.66)	0.22	0.16 (0.02, 1.64)	0.12
Hours greater than 75 $\mu g/m^3$ $PM_{2.5}$						
≤ Median (11.575hours)	17 (63)	15 (56)	REF		REF	
> Median	10 (37)	12 (44)	2.00 (0.37, 10.92)	0.42	5.27 (0.34, 81.25)	0.23
Hours greater than 75 $\mu g/m^3$ $PM_{2.5}$, median (IQR)	7.1 (4.5, 18.1)	11.0 (4.1, 16.1)	1.00 (0.88, 1.13)	0.94	1.00 (0..83, 1.19)	0.96
Hours greater than 75 $\mu g/m^3$ $PM_{2.5}$, quartile						
1 [0.183,5.44]	10 (37)	9 (33)	REF		REF	
2 (5.44,11.6]	7 (26)	6 (22)	1.50 (0.32, 7.12)	0.61	1.06 (0.16, 7.28)	0.95
3 (11.6,18.8]	4 (15)	9 (33)	3.84 (0.40, 36.55)	0.24	5.68 (0.18, 176.45)	0.32
4 (18.8,24]	6 (22)	3 (11)	0.67 (0.04, 10.37)	0.77	2.54 (0.03, 221.39)	0.68

Hours greater than 75 μg/m^3 $PM_{2.5}$, quartile (continuous)	2.0 (1.0, 3.0)	2.0 (1.0, 3.0)	1.00 (0.45, 2.23)	1.0	1.33 (0.36, 4.90)	0.67
Hours greater than 75 μg/m^3 $PM_{2.5}$ for LPG as primary cook fuel						
≤ Median (11.65 hours)	18 (67)	15 (56)	REF		REF	
> Median	9 (33)	12 (44)	4.00 (0.45, 35.79)	0.22	16.98 (0.49, 585.66)	0.12
Hours greater than 75 μg/m^3 $PM_{2.5}$ for LPG as only cooking fuel						
≤ Median (11.65 hours)	18 (67)	15 (56)	REF		REF	
> Median	9 (33)	12 (44)	4.00 (0.45, 35.79)	0.22	16.98 (0.49, 585.66)	0.12

***** Bolded values statistically significant at $p < 0.05$

¥ Multivariate analysis controlling for Principal Component 1 Score above or below the median value, Principal Component 3 Score above or below the median value, having a separate cooking space, always opening the door when cooking, always keeping a window open when cooking, and crowding

Supplemental Tables

Table 2.S.1. Dichotomized scores resulting from principal component analysis with promax rotation for households of adult women and child index TB case participants and their matched controls (n=118 individuals, n=59 pairs) in Pune, India.

	Control (n=59)	**Case (n=59)**	**Univariate OR (95% CI)**	**p-value***
Principal Component Score 1, n(%)				
< Median	33 (56)	26 (44)	REF	
≥ Median	26 (44)	33 (56)	2.4 (0.85, 6.81)	0.10
Principal Component Score 2, n(%)				
< Median	33 (56)	26 (44)	REF	
≥ Median	26 (44)	33 (56)	0.55 (0.20, 1.47)	0.23
Principal Component Score 3, n(%)				
< Median	24 (41)	35 (59)	REF	
≥ Median	35 (59)	24 (41)	**0.45 (0.21, 0.99)**	**0.05**

* Bolded values statistically significant at $p < 0.05$

Table 2.S.2. Principal component analysis pattern matrix for socioeconomic status among households of adult women and child index TB case participants and their matched controls (n=118 individuals, n=59 pairs) in Pune, India.

	PC1 Lambda	**PC2 Lambda**	**PC3 Lambda**	**Communality**	**Uniqueness**	**Complexity**
Number of people in the home (quartile)	0.17	0.81	0.11	0.61	0.392	1.1
Years lived in the home (quartile)	-0.03	0.81	-0.13	0.67	0.326	1.1
Ownership of other land	0.48	-0.35	0.71	0.71	0.287	2.3
Joint or extended family (vs nuclear)	0.22	0.94	0.17	0.82	0.176	1.2
Income greater than 10,000 INR	-0.14	0.13	0.68	0.59	0.409	1.2
Reported food insecurity	-0.03	-0.06	-0.79	0.61	0.389	1.0
Ownership of watch	-0.72	0.18	-0.14	0.58	0.421	1.2
Ownership of chair	-0.53	-0.01	0.44	0.64	0.363	1.9
Ownership of refrigerator	-0.22	0.52	0.44	0.69	0.314	2.3
Ownership of motorcycle	-0.41	0.05	0.53	0.62	0.379	1.9
Ownership of pressure cooker	-0.71	0.22	-0.05	0.62	0.378	1.2
Home not owned	-0.11	-0.92	-0.01	0.79	0.206	1.0
Roofing material plastic, rudimentary, or metal sheet	0.88	0.31	-0.08	0.74	0.256	1.3
Exterior walls made of bamboo/mud or metal sheets	0.97	0.08	-0.02	0.90	0.095	1.0
Flooring concrete or rudimentary (no tiles)	0.86	-0.01	0.06	0.71	0.289	1.0
Housing condition in need of repairs	0.86	0.32	-0.26	0.87	0.128	1.5
Has health insurance	0.00	0.11	0.71	0.52	0.476	1.0

Table 2.S.3. Proportion of variance explained by principal components of socioeconomic status among households of adult women and child index TB case participants and their matched controls (n=118 individuals, n=59 pairs) in Pune, India.

	PC1	**PC2**	**PC3**
SS Loadings (Eigen Values)	5.02	3.64	3.06
Proportion Variance Explained	0.30	0.21	0.18
Cumulative Variance Explained	0.30	0.51	0.69
Proportion Explained of PCs	0.43	0.31	0.26
Cumulative Proportion of PCs	0.43	0.74	1.00

Table 2.S.4. Variables strongly loaded (lambda was ≥ |0.5|) on principal component results from principal component analysis with promax rotation for socioeconomic status among households of adult women and child index TB case participants and their matched controls (n=118 individuals, n=59 pairs) in Pune, India.

PC1	PC2	PC3
Ownership of watch	Number of people in the home (quartile)	Ownership of other land
Ownership of chair	Years lived in the home (quartile)	Income greater than 10,000 INR
Ownership of pressure cooker	Joint or extended family (vs nuclear)	Reported food insecurity
Roofing material plastic, rudimentary, or metal sheet	Ownership of refrigerator	Ownership of motorcycle
Exterior walls made of bamboo/mud or metal sheets	Home not owned	Has health insurance
Flooring concrete or rudimentary (no tiles)		
Housing condition in need of repairs		

Table 2.S.5. Structure matrix of correlations between variables and principal components resulting from principal component analysis with promax rotation for socioeconomic status among households of adult women and child index TB case participants and their matched controls (n=118 individuals, n=59 pairs) in Pune, India.

	PC1	PC2	PC3
Number of people in the home (quartile)	-0.13	0.76	0.10
Years lived in the home (quartile)	-0.24	0.81	-0.08
Ownership of other land	0.35	-0.47	0.53
Joint or extended family (vs nuclear)	-0.14	0.88	0.14
Income greater than 10,000 INR	-0.41	0.21	0.74
Reported food insecurity	0.26	-0.10	-0.78
Ownership of watch	-0.73	0.41	0.11
Ownership of chair	-0.68	0.19	0.62
Ownership of refrigerator	-0.54	0.62	0.54
Ownership of motorcycle	-0.61	0.21	0.67
Ownership of pressure cooker	-0.76	0.44	0.20
Home not owned	0.19	-0.89	-0.02
Roofing material plastic, rudimentary, or metal sheet	0.81	0.02	-0.36
Exterior walls made of bamboo/mud or metal sheets	0.95	-0.23	-0.34
Flooring concrete or rudimentary (no tiles)	0.84	-0.28	-0.23
Housing condition in need of repairs	0.84	0.03	-0.53
Has health insurance	-0.28	0.15	0.72

Table 2.S.6. Factor correlation matrix for principal components resulting from principal component analysis with promax rotation for socioeconomic status among households of adult women and child index TB case participants and their matched controls (n=118 individuals, n=59 pairs) in Pune, India.

	PC1	**PC2**	**PC3**
PC1	1.00	-0.32	-0.34
PC2	-0.32	1.00	0.05
PC3	-0.34	0.05	1.00

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CHAPTER 3

Household air pollution, fuel use patterns, and the correlation between fine particulate matter and carbon monoxide in low-income urban Indian homes

Abstract

Background: Household air pollution (HAP) is accountable for over 3.5 million annual deaths. Characterization of exposure and determinants of HAP inform intervention strategies, and this has been poorly characterized in low-income urban Indian communities.

Methods: Household measurements of HAP were collected from homes in low-income urban Indian communities. A structured questionnaire was administered to assess participant-reported sources of HAP, including cooking fuel, incense, mosquito coils, trash burning, and secondhand tobacco smoke (SHS). Household concentrations of $PM_{2.5}$ and CO were collected using direct-reading instruments for a period of 24 hours in each home.

Results: Kerosene and biomass use were prevalent in these communities (22% and 27%, respectively), mostly as secondary fuel sources. LPG alone was used by 61 (51%) of homes. Kerosene was primarily used inside the home (n=26, 93%), while wood was primarily used outside of the home (n=25, 86%). High concentrations of 24-hour $PM_{2.4}$ (184 $\mu g/m^3$; IQR: 113, 347) were observed in all homes, regardless of cooking fuel source. Those in the lowest quartile of exposure had a great majority of monitoring time lower than the WHO interim target of 75 $\mu g/m^3$. Households in the highest quartile of exposure tended to use wood as a cooking fuel (OR 5.22; 95% CI: 1.46, 20.05) and use mosquito coils (OR 4.47; 95% CI: 1.56, 15.38). Low correlation was seen between measures of $PM_{2.5}$ and CO, likely due to the high prevalence of multiple exposure sources in this population.

Discussion: Low-income communities in urban India are highly exposed to HAP, even when only using LPG as a fuel source. Low- and high-exposure populations were identified, and those with the highest exposure tended to use wood as a cooking fuel and use mosquito coils. Interventions addressing these exposures are needed, and will likely be most efficient at the neighborhood, as opposed to household, level in these types of communities.

Introduction

Household air pollution (HAP) is an established risk factor for respiratory disease in adults and children, and is of particular concern in low- and middle-income countries (LMIC) where biomass fuels are used for cooking and heating. It is estimated that exposure to air pollution lead to 4.3 million deaths in 2012, more than 3.5 million of which were from HAP[1]. The vast majority of published literature characterizing HAP exposures, as well as the household conditions in which they occur, is from biomass sources in predominantly rural areas of LMIC[2-8]. Rural communities largely rely on biomass for cooking, and often do not have access to liquid petroleum gas (LPG) or clean electric sources, either due to availability or financial restrictions. Those living in urban settings often have higher access to these cleaner fuels, however they may still rely on unclean fuels such as wood and kerosene for cooking, heating, and lighting[9]. Current evidence suggests that HAP in urban areas is also extremely high, and that characteristics unique to urban settings may play an important role in exposure, however HAP in densely populated urban environments of LMICs have not been sufficiently characterized to provide strong evidence for effective intervention strategies, as many of the studies in urban areas include a limited number of households in their analysis[10-17].

Detailed information on levels of HAP is required to design well-informed strategies to reduce exposure and subsequent burden of disease[18]. While outdoor air pollution is an important exposure for those in urban environments, the household level represents low-hanging fruit in terms of intervention strategies. Concern over outdoor air pollution in these areas may overshadow meaningful exposures and potential interventions at the household level. Urban communities may also have a unique set of risk factors as compared to rural settings[17]. Identification of modifiable risk factors that are most easily targeted for intervention is an important first step in improving indoor air quality and lowering exposure to harmful combustion

products. Identifying household characteristics associated with higher levels of HAP can be used to potentially inform low-cost and simple intervention strategies to reduce exposure, however it is important to understand the context of the heterogeneous environments in which people live. For example, simple strategies such as opening windows or moving cooking fuel out doors have been suggested in some peri-urban and rural settings[4, 8]. Ventilation in low-income urban communities, however, may look very different than those in rural areas[19] . The pollution sources across these different settings, such as the use of incense, mosquito coils, candles, and domestic burning of trash, all which release $PM_{2.5}$ and other pollutants, are an important source of variability[20, 21]. Dust, road traffic, and other ambient air sources have also been found to penetrate into indoor environments[14].

With greater access to resources and fewer options for inexpensive or free biomass fuel, multiple sources of fuel are often found in urban settings[11, 14]. Concern about the health impacts of kerosene stoves begs additional research, as kerosene is not well characterized as a cooking fuel, and understanding the contribution of kerosene stoves to traditionally measured pollutants, such as $PM_{2.5}$ and carbon monoxide (CO), is important[22]. Further, many studies compare the use of wood-burning fuels with kerosene in the clean fuel category, when kerosene combustion produces toxic components of concerns[22].

In addition to informing interventions, research is needed to understand how to best measure HAP exposures across various populations and develop appropriate strategies for measuring impact of exposure reduction strategies. Reported measures of exposure to pollutants in the home, such as type of fuel used, use of mosquito coils, and domestic burning of trash, may not accurately classify household exposures. Air monitoring strategies, such as quantifying concentrations of $PM_{2.5}$, provide more detailed and precise information on exposure patterns in

the home, but are expensive and require careful technical administration. CO measurements are often substituted as a proxy for $PM_{2.5}$ due to the ease of collection and inexpensive monitoring options. Strong correlations have been found in kitchens using biomass smoke for cooking between $PM_{2.5}$ and CO, suggesting that in some settings this may be an appropriate strategy[23, 24]. In settings where there is a dominant source of biomass combustion, personal CO has been found to be a reliable surrogate for personal PM[5]. Other studies have suggested that CO is not a good substitute for $PM_{2.5}$ and does not fully explain variability in exposure[4, 25]. In rural settings, fuel use patterns, housing conditions, and type of cook stove have been shown to modify exposure to indoor pollutants. The housing conditions and structures of urban households, especially those in low-income areas, provide a unique set of conditions that likely contribute to indoor air quality[19]. Characterization of these conditions in combination with objective pollutant measurements is needed to determine what type of error might be introduced into studies by neglecting to capture additional descriptive information on housing characteristics. Additionally, variation in cooking behavior and placement of the stove and monitor may have implications for measurement.

In the present study we aim to characterize household air pollution patterns in low-income urban Indian households. We also explore associations between modifiable risk factors at the household level and concentrations of $PM_{2.5}$ and CO, as well as assess if CO is a reliable surrogate for $PM_{2.5}$ concentration in these homes.

Methods

Ethics approval for this study was granted from the Institutional Review Boards of the Sassoon General Hospital and Byramjee Jeejeebhoy Medial College (SGH/BJMC) in Pune, Maharashtra,

India and the Johns Hopkins University School of Medicine in Baltimore, Maryland, USA. All participants provided written informed consent for participation.

Study Population

Pune is a large district located in Maharasthra, a state in the western region of India. The district has a population of nearly 9.5 million, living in both urban (5.7 million) and rural (3.7 million) areas[26]. Research was conducted in partnership with Sassoon General Hospital (SGH) – Byramjee Jeejeebhoy Medical College (SGH/BJMC), Pune, India. BJMC is a large Maharashtra Government tertiary care public and teaching hospital, primarily serving the lower socioeconomic communities in Pune and surrounding areas. Participating households in this study were recruited as part of on-going research to assess the association between HAP and tuberculosis (TB) (parent study), and are populations served by SGH/BJMC.

Exposure Assessment

Exposure to air pollution from cooking fuels, secondhand tobacco smoke (SHS), and other pollution sources was measured in all participating households using structured questionnaires and objective measures of $PM_{2.5}$, CO, and air nicotine.

All questionnaires were translated into Marathi in order to ensure that subjective questions would be asked in a standardized way, with response options that were clear to the participants, and to ensure that they were true to their intention. Questionnaires were first translated into Marathi and back-translated into English. Discrepancies were identified, and further edits were made by study team consensus. Questionnaires assessing patterns of exposure both in the home and outside the home were administered to each participant at the baseline visit. Participants were asked about typical use and exposure over the past 7 days regarding the types of fuel used in the home, what

the fuel was used to heat or cook, and the duration of use of these fuels. Information on ventilation was collected by asking participants whether they opened doors or windows when cooking. Additional reported exposures collected included trash burning near the home, exposure to neighbors using wood for cooking or regular preparation of Mishri (a smokeless tobacco product prepared by burning). Participants also reported the use of mosquito coils, incense, and candles or kerosene for lighting. Details of the housing construction and ventilation were recorded by observation. This included information on household characteristics such as construction materials of the walls, roof, and floor, the presence of a gap between the ceiling and the roof, the presence of a separate kitchen, and the size of the cooking space.

Household $PM_{2.5}$ was assessed using the Thermo Environmental Instruments pDR-1000 sampler (Thermo Fisher Scientific, Waltham, MA) fitted with a cyclone inlet (BGI, Waltham, MA) and paired with portable constant-flow pumps (SKC Inc, PA). Pumps were pre- and post-calibrated at 4 L/min using a Bios DryCal primary flow calibrator (MesaLabs, Lakewood CO). A pre-weighed Teflon filter was placed downstream in pre-loaded cassettes for gravimetric measurements. Lascar direct-reading CO monitors (EL-CO-USB 300, Lascar Electronics, Erie, PA) were also paired with the monitoring set-up, which was placed approximately 1 x 1 meter away from the primary cook stove in each home. Nephelometric measurements of $PM_{2.5}$ and CO concentration were collected every minute for a period of 24 hours. Filters were post-weighed to assess accumulation of particulate matter over the sampling period. Mean exposure comparing nephelometric measurements to gravimetric measurements was used to create a calibration factor that was applied to each nephelometric measurement prior to analysis[27]. Blank filters were included for quality control purposes at 10% of the home visits. Field counselors brought these blank filters into the field, opened and closed the filters in the study location, and indicated on the data collection forms the cassette number and that it was a field blank.

Passive samplers for vapor-phase nicotine were used for the quantification of secondhand smoke in the home. The research team placed one air nicotine monitor in the home, which was left in place for a period of seven days. For quality control purposes, a 10% sample of blanks and a 10% sample of duplicates were included. Samples were analyzed at the JHSPH Secondhand Smoke Exposure Assessment Laboratory in Baltimore, MD, USA.

Statistical Analysis

Gravimetric-equivalent concentrations for nephelometric measures were calculated using down-stream filter concentrations as a standard[27]. For nephelometric measurements less than the limit of detection (0.001 mg/m^3), a correction of the LOD / square root of 2 was applied. Mean 24-hour concentrations for $PM_{2.5}$ and CO were calculated for each home. Additionally, 15-minute moving averages were calculated for CO and nephelometric data for comparison with the 15 minute moving average limits for CO as outlined by the World Health Organization[28]. Households were also categorized into quartiles by 24-hour mean $PM_{2.5}$ concentrations. The number of hours above 75 $\mu g/m^3$ $PM_{2.5}$ was also calculated for each home in an effort to understand peak measures of exposure, and this threshold was chosen as it is an interim target as described by WHO standards for air pollution exposure[29]. Air nicotine concentrations were first categorized as undetectable and detectable. The median value of detectable nicotine was then calculated and a new variable created classifying individuals as undetectable, low detectable, and high detectable. Median and interquartile range values were calculated for all continuous measures of HAP, and all measures of HAP were compared across fuel types (primary and composite) using the Kruskal-Wallis test. A composite variable for fuel type was calculated to account for the use of multiple fuels, categorizing households as using LPG only, kerosene but no biomass, and any biomass use.

Linear regression was performed with log-transformed 24-hour averages of $PM_{2.5}$ and CO and our exposures of interest. Additional logistic regression models were built to consider the associations of reported exposures of interest with those above the median number of hours of $PM_{2.5} > 75$ $\mu g/m^3$, those in the highest three exposure quartiles for mean 24-hour $PM_{2.5}$, and those in the highest exposure quartile for mean 24-hour $PM_{2.5}$. Multiviarate regression models for these outcomes of interest were built with fuel variables as the primary exposure of interest, controlling for covariates found to be significant in univariate analysis with $p<0.10$, or considered to be of epidemiologic importance *a priori*. Correlations between mean 24-hour $PM_{2.5}$ and CO concentrations were calculated using the Spearman Rank test for all households together, then in separate categories by primary cooking fuel and composite cooking fuel variable.

Results

A total of 127 households were enrolled, of which eight (6%) were excluded from this analysis due to problems with air monitoring data. Of the remaining 119 homes included, all have complete measures of 24-hour continuous $PM_{2.5}$ readings, and 117 (98%) have complete measures of CO paired at the primary cook stove. Descriptive statistics about cooking fuel are presented in Table 3.1. The majority of households reported using LPG as their primary fuel source (n=101, 85%), and a lesser proportion reported kerosene (n=14, 12%) and wood (n=4, 3%) as the primary cooking fuel. Over 40% of homes, however, reported a secondary fuel source used on a regular basis. Wood was predominantly reported as this secondary fuel source (n=28, 24%), however electricity (n=7, 6%) and kerosene (n=16, 13%) were also reported. A composite variable was made to capture this diversity of fuels used in the home, and 61 (51%) participants used LPG only, 26 (22%) used some kerosene but no wood, and 32 (27%) participants reported

using some wood, either as the primary or secondary fuel source. No households reported biomass use other than wood. Participants reported cooking more frequently and for the longest periods of time in the early morning and at dinner time, although some cooking or heating of food or water was reported during all times of the day. In total, participants reported cooking a median of 135 minutes (IQR: 93 – 195) each day.

All cooking with LPG, and most cooking with kerosene, happens inside of the home (n=101, 100% and n=26, 93%, respectively) as compared to an outside cooking area. While indoor environments predominated as the cooking space for LPG and kerosene, wood fuel is primarily used outside of the home (n=25, 85%). When asked about opening windows when cooking, only 66 (55%) reported always or nearly always opening them during cooking. The remaining opened them less than half of the time or never, and 38 (32%) did not have windows in their cooking area leading to outsie spaces. A larger proportion reported opening kitchen doors more than half of the time while cooking (n=105, 88%).

Reported exposure to other household pollutants is shown in Table 3.3. Weekly or daily burning of trash by the participant or their neighbors was reported by 26 (22%) homes. Incense is used by 102 (85%) participants a median of 15 minutes (IQR: 10 – 24) per day. Mosquito coils, used by 30 (25%) of the participants, were used for much longer periods of time each day at a median of 450 minutes (IQR: 60 – 600). In addition to electricity, reported light sources included kerosene (n=13, 11%) and candles (n=82, 69%). A subset of participants was asked about exposures to pollution originating from other households, and 36% reported smelling others use biomass every day.

High concentrations of monitored air pollutants were found in homes, which are summarized in Table 3.4. The overall median 24-hour average concentration of $PM_{2.5}$ was 184 $\mu g/m^3$ (IQR: 113 – 347), more than seven times higher than the WHO recommended maximum exposure level of 25 $\mu g/m^3$ over a 24-hour period. Although there was not a statistically different difference between the three categories of primary cooking fuel, the median values of 24-hour average $PM_{2.5}$ was statistically significantly different between the categories of composite cooking fuel, with those using wood or kerosene as a secondary source tending to have a higher average concentration than those using LPG alone. We calculated the number of hours where the concentration of $PM_{2.5}$ was above the WHO interim target of 75 $\mu g/m^3$. Overall, households were above the 75 $\mu g/m^3$ threshold for a median of 11.6 hours of each day. Similar to the 24-hour average values, differences in exposure tended to be seen when homes were categorized by the composite fuel variable. Unlike measures of $PM_{2.5}$, 24-hour averages of CO were significantly different across the fuel types for both categorization methods. Graphical depictions of exposure levels are provided in Figures 3.1 – 3.6.

Univariate and multivariate linear regression analyses were conducted to assess cooking fuel and kitchen characteristics with log-transformed 24-hour average $PM_{2.5}$ and CO measures. Univariate analysis for average 24-hour $PM_{2.5}$ and CO concentrations are presented in Table 3.5. In analyses for primary cooking fuel, observations with wood as the primary fuel source were removed due to the low number in that category. Significant associations were found between the any biomass use composite category and $PM_{2.5}$ (Coef: 0.74; 95% CI: 0.28 – 1.20) and between kerosene use composite category and CO (Coef: 0.49; 95% CI: 0.08, 0.90) in univariate analysis. Other household predictors associated with $PM_{2.5}$ in univariate analysis included all concrete or brick construction material of the home and whether or not there was a visible gap between the walls and the ceiling, although it is worth noting that these variables are highly correlated. Incense was

found to be negatively associated with $PM_{2.5}$, indicating there may be an association between incense and our pollutant outcome that is unaccounted for as this direction of association is opposite of what we would anticipate. No other household predictors were found to be significantly associated with $PM_{2.5}$, and no household predictors were significantly associated with CO levels. Measurements taken in the winter season, however, were significantly associated with our outcomes of interest.

In multivariate regression, kitchen areas made of all concrete or brick were significantly associated with lower levels of average $PM_{2.5}$ (Table 3.6), as were measurements taken in the winter season (Coef: 0.47; 95% CI: 0.05, 0.89), however no other variables, including cooking fuel, reached a statistically significant association. In multivariate analysis comparing characteristics across average CO concentrations, our model including composite score of cooking fuel for the restricted data set showed kitchens with some concrete or brick constructions being marginally associated with increased concentrations of CO. Kerosene use was also statistically significantly associated with increased CO concentrations in models including composite scores of cooking fuels, and approached statistical significance when comparing kerosene as the primary cooking fuel to LPG as the primary cooking fuel. All models showed a positive and statistically significant association with measurements in the winter months (Table 3.7).

In the parent project of this study, the relationship between number of hours that $PM_{2.5}$ was greater than 75 $\mu g/m^3$ and our outcome of interest was approaching a statistically significant association. In the present study, we found that households with cooking spaces made of all concrete or block had decreased odds of air pollution concentrations greater than this threshold (OR: 0.34; 95% CI: 0.12 – 0.88). Households with larger cooking spaces (defined as having a

kitchen volume greater than the median) also had significantly decreased odds of exposure in univariate analysis (OR: 0.37, 0.17 – 0.77). In controlled analysis, however, only construction material was significantly associated with our outcome, resulting in decreased odds of exposure for kitchens made of all concrete or brick as compared to those made of all corrugated metal (Table 3.9). Additionally, sampling during the winter season was positively associated with our outcomes.

Figure 3.6 provides a graphical display of minute-to-minute median $PM_{2.5}$ concentrations by quartile of 24-hour averages. The red and orange dashed lines depict the WHO standards of 25 $\mu g/m^3$ and the interim target of 75 $\mu g/m^3$ for a 24-hour period of time, respectively. It is worth noting that households in the lowest quartile of exposure have median values below the interim target level for nearly the entirety of the monitoring period. In univariate logistic regression (Table 3.9), the use of any biomass fuel in the composite variable was positively and significantly associated with being in the upper three exposure quartiles (OR 3.95; 95% CI: 1.33, 14.63) as compared to being in the bottom exposure quartile, and always opening a window during cooking activities significantly decreased odds of being categorized as high exposure (OR: 0.43; 95% CI: 0.18 – 1.00). In multivariate analysis, however, only winter season for the restricted model of using composite cooking fuel was significantly associated with being above the first exposure quartile (Table 3.10).

We next investigated household characteristics associated with being in the highest exposure quartile, as these individuals represent those at highest exposure and likely the greatest risk of subsequent disease. Univariate measures of association are presented in Table 3.11, and adjusted multivariate analyses for odds of being in the top exposure quartile are presented in Table 3.12. Similar to other models, concrete and brick construction materials, as well as season of

measurement, was positively and significantly associated with the outcome. Of note, however, is that the reported use of mosquito coils was also strongly positively and significantly associated with greatest exposure in all three regression models.

A scatterplot of the correlation between 24-hour concentrations of $PM_{2.5}$ and CO for all households combined is presented in Figure 3.14, where a moderate correlation between the two measurements is seen ($r = 0.45$, $p<0.0001$). When categorizing by primary cooking fuel (Figure 3.15), CO is moderately and significantly correlated with $PM_{2.5}$ among homes using LPG, but not associated for kerosene ($r = 0.32$, $p = 0.28$) or wood ($r = 0.8$, $p=0.33$), although only four observations are present for the latter association. Correlations by composite cooking fuel exposure show a positive and moderate association for those using LPG only ($r = 0.58$, $p < 0.0001$) and for households using any wood ($r = 0.51$, $p=0.004$), however not for households in the kerosene category ($r = 0.17$, $p=0.40$).

Discussion

This study assessed reported measures of exposure to HAP and concentrations of $PM_{2.5}$ and CO over a 24-hour period of time in low-income households of urban India. While a majority of participants reported using LPG as their primary cooking fuel, duel-fuel use was highly prevalent. Concentrations of 24-hour $PM_{2.5}$ were well over the WHO upper limit of 25 $\mu g/m^3$, regardless of fuel type. Housing structure significantly decreased exposure to $PM_{2.5}$ in adjusted analysis, and across all analyses, measurements taken in the winter months showed a positive relationship with HAP. CO, on the other hand, was not associated with housing structure, but higher levels of CO were seen among those using kerosene as compared to LPG. No statistically significant increases in $PM_{2.5}$ or CO measures were seen in linear regression among those using wood, likely because

wood was primarily used outside of the home and measurements were collected indoors. Additionally, CO was not strongly correlated with $PM_{2.5}$ measurements, and is likely not a good proxy for combustion generated air pollution exposure in this setting.

We assessed several different aspects of kitchen space in relation to concentration of air pollutants found in the home, and different aspects of the cooking space were found to be important for different measures of pollutant. Kitchen areas with construction material of all concrete or brick had lower concentrations of continuous 24-hour $PM_{2.5}$ than did homes constructed with any corrugated metal. It is likely that this aspect of the housing structure plays an important role in keeping wood-based combustion products, as well as ambient air pollution, out of the home. Other studies show mixed results with regard to construction materials and variability in pollutant concentrations. In Honduran homes primarily using wood for cooking fuel, Clark et al. report housing construction materials do explain variability in $PM_{2.5}$ and CO concentrations. Klasen et al. found that in rural Peru, Nepal, and Kenya, construction materials of the wall or roofs was not associated with concentration of indoor pollutants. They did, however, find that having at least two windows (vs fewer) was significantly associated with decreases in $PM_{2.5}$. We did not find that opening windows or doors mitigated HAP in our participating homes. The rural locations in the Klasen study, as compared to the urban homes of our study, could potentially explain these differences. In another study of wood-burning households, McCracken et al. reported that kitchen measures were not a good marker of exposure in the home[5].

We found that variability in only CO and number of hours of $PM_{2.5}$ above 75 $\mu g/m^3$ could be explained by reported fuel use, likely due to a combination of location and duration of use of kerosene and wood. It should be noted, however, that the multiple sources of fuel used in the communities from which we recruited makes it difficult to compare to studies among populations

primarily using wood and other biomass fuel. In Honduran homes, cooking stove and fuels used in homes were found to explain variability in $PM_{2.5}$ and CO concentrations in homes, however all included homes used wood-burning stove (either improved or traditional), whereas duel-use of both clean and unclean fuels is quite prevalent in our population[3]. Klasen et al. reported exposures in rural Peru, Nepal, and Kenya, among which 20% of households reported secondary fuel use of LPG or kerosene. Due to primary use of wood fuels in these rural settings, concentrations of HAP were much higher than in the present study, and ever-use of an LPG stove did not significantly decrease HAP levels in multivariate analysis[8]. A study among low-income urban households in Bangladesh, a setting more similar to that of urban India and one where 64% of households used LPG as their primary cooking fuel, found that indoor $PM_{2.5}$ concentrations were associated with ventilation of the home, defined by the number of external windows and doors[16].

Notably, the reported use of mosquito coils was strongly and positively associated with being classified in the highest quartile of 24-hour average $PM_{2.5}$. Very few studies have investigated the contribution of mosquito coil emissions with household levels of $PM_{2.5}$ in homes. While most emission analyses have been conducted in the laboratory, in a controlled setting in an Indian home, the burning of a mosquito coil was found to produce a mean $PM_{2.5}$ concentrations of 1031 $\mu g/m^3$ and mean CO concentration of 6.5 ppm[21]. Laboratory studies indicate that the $PM_{2.5}$ mass product from burning one mosquito coil is equal to that of the burning of 75-137 cigarettes, all the while containing additional dangerous chemical constituents[30]. To our knowledge, no other published study has assessed the contribution of mosquito coils to $PM_{2.5}$ mass in uncontrolled household monitoring. This exposure represents one of the most immediate opportunities for intervention to reduce HAP among the most highly exposed individuals in this population.

Few studies have been conducted in low-income urban areas of India. Saksena et al. measured HAP in low-income settlements in Delhi, focusing on communities that primarily used wood for cooking. Among the 80 homes included in this study, both wood and kerosene were found to be used, wood both inside and outside the home, and kerosene primarily inside of the home. Not surprisingly, households using wood had higher concentrations of particulates than kerosene households, however those using kerosene and categorized as "low-pollution" communities still had geometric mean PM_5 levels of approximately 600 $\mu g/m^3$ [17]. A second study in low-income areas of Dehli attributed high indoor measures of $PM_{2.5}$ to poor ventilation. The authors only present prevalence of fuel use for LPG and kerosene, although it is mentioned that wood is also used in this population. Nevertheless, levels of $PM_{2.5}$ are also highly elevated, especially in the winter season. Variation of HAP is presented in this study by season and housing characteristics, however variation by fuel use or other pollutant sources are poorly defined[12]. In urban homes in Agra, India, 24-hour concentrations of $PM_{2.5}$ were similar to the results we present, and both indoor and outdoor activities were found to increase exposure, however only a small number of homes were monitored[31].

The low levels of correlation between measures of $PM_{2.5}$ and CO in the households monitored is consistent with studies evaluating households with mixed fuel types. The majority of studies that conclude CO is as adequate a marker of combustion as $PM_{2.5}$ occur in households exclusively using wood for cooking[5, 23]. Even so, studies where wood is the dominant fuel source find low correlation between these two pollutants[4]. While $PM_{2.5}$ is generally a more complicated and expensive measurement to take, evidence suggests that in settings such as low-income urban Indian communities where multiple fuel sources are used, $PM_{2.5}$ measurements should be prioritized.

Due to logistic and financial restrictions, we were limited to collecting household measurements at one time point instead of multiple measurements across seasons in each home. This would have allowed us to account for not only day-to-day variability in the households we recruited, but also by season. We did, however, enroll and sample homes throughout the year, and were able to control for seasonal variations in multivariable models. Additionally, our method for controlling for ventilation factors were limited to observable characteristics of the home and reported ventilation activities by the participants. These proxy measures likely do not capture the full variability in ventilation of the included homes, however do provide insight into potential modifications at the household level that could increase ventilation.

An important strength of this study is the use of direct-reading instruments for data collection and our subsequent ability to associate kitchen characteristics with hours over the 75 $\mu g/m^3$ threshold interim target from the WHO. In the parent study, our findings suggest that number of hours with a concentration greater than 75 $\mu g/m^3$ is positively associated with TB in adult women and young children. Understanding contributing household characteristics associated with these high concentrations of $PM_{2.5}$ is important for understanding context and planning intervention strategies and future studies. In the parent study of this project, reported kerosene use is also positively associated with our primary outcome of interest (TB), however the use of wood fuel is not. Here we report that measurements of $PM_{2.5}$, our primary exposure of interest, do not appreciably vary between households reporting LPG only and those reporting any kerosene use. $PM_{2.5}$ levels produced by the combustion of kerosene are much lower than that of wood, however ultrafine particles produced by kerosene may not significantly contribute to mass measurements of exposure. Future studies are needed to characterize ultrafine particle emissions from these stoves, as well as gaseous and other chemical components of kerosene combustion, which may play an important role in human disease. Additionally, our study is one of the first to characterize

the contribution of mosquito coil combustion products to particulate mass in an uncontrolled setting.

Additional research is needed to understand characteristics that separate those in the lowest-exposure categories from those in the highest-exposure categories. While we have identified several contributing factors, additional indicators, such as the contribution of ambient air pollution to indoor values in this setting, will better inform strategies for exposure reduction. Interim strategies to transition households from corrugated metal construction materials to concrete or brick may be financially unfeasible or logistically challenging, however our results indicate that decreasing wood use as a secondary fuel source could have positive impact on indoor air quality. Further, households should consider no longer using mosquito coils as a repellent, as they are positively associated with high levels of pollution. The benefit of decreased air pollution exposure, however, will have to be weighed against the potential for increased exposure to mosquito-borne illness. Future studies should assess the true effectiveness of mosquito coils to prevent against vector-borne diseases, and the alternate scenario of benefit from better air quality. Any intervention employed in this type of setting should consider implementation at the neighborhood level, as interventions at only the household level may fail to reduce exposure from other homes in these densely populated communities. For example, Akunne et al (2006) found that if families primarily using wood-burning stoves shifted their cooking practices outside, ARI in children < 5 years could be reduced by at least 50%. This study was done, however, in a less-densely populated area of Burkina Faso[8]. In a low-income urban India setting, with households densely packed, simply moving a cooking stove outside may not appreciably reduce exposure to both the household using as well as other neighboring homes.

Our study shows a subset of homes that fall into the lowest quartile of exposure are reaching the interim target for 24-hour exposure, as well as distinguishing characteristics of the subset of homes falling in the highest exposure quartile. Additional research is needed to understand the determinants of exposure and potential interventions that could help us move those in the highest quartiles to where the lowest quartile is. Further, we have identified characteristics of those most exposed, including secondary use of wood fuel and use of mosquito coils, which begs further research on the contribution of these exposures to disease in these populations, as well as implementation research on strategies for mitigating exposure. While levels of air pollution remain so high, ongoing research is needed to understand the climate of exposure, levels of risk, and opportunities for intervention in these highly vulnerable, and highly exposed, populations.

Table 3.1. Reported use of cooking fuel use and cooking behaviors in low-income urban Indian households (n = 119).

	n=119
Primary Fuel Types, n(%)	
LPG or electric	101 (85)
Kerosene	14 (12)
Wood	4 (3)
Secondary Fuel Types, n(%)	
None	68 (57)
Electricity	7 (6)
LPG	0 (--)
Kerosene	16 (13)
Wood	28 (24)
Composite Fuel Types†, n(%)	
LPG/electricity only	61 (51)
Kerosene (no biomass)	26 (22)
Any biomass	32 (27)
Times of day usually cook (7 day), n(%)	
Early morning	117 (98)
Between morning and lunchtime	73 (61)
Lunchtime	59 (50)
Between lunch and dinner	90 (76)
Dinner	117 (98)
Late evening	22 (18)
Amount of time cook in minutes (among those reporting cooking), median (IQR)	
Early morning, median (IQR)	70 (30, 120)
Between morning and lunchtime	60 (25, 90)
Lunchtime	15 (10, 15)
Between lunch and dinner	15 (10, 20)
Dinner	60 (60, 90)
Late evening	0 (0, 0)
Amount of time cook in minutes (among all), median (IQR)	
Early morning	60 (30, 120)
Between morning and lunchtime	15 (0, 60)
Lunchtime	5 (0, 15)
Between lunch and dinner	15 (5, 15
Dinner	60 (60, 90)
Late evening	0 (0, 0)
Total time cooking in minutes, median (IQR)	135 (93, 195)
Use of primary cook fuel per day (7 day), n(%)	
Less than 30 minutes	1 (1)
30 minutes to 1 hour	2 (2)
Between 1-2 hours	8 (7)
More than 2 to 3 hours	48 (40)
More than 3 to 5 hours	51 (43)
More than 5 hours	9 (8)
Use of secondary cook fuel per day (7 day), n(%)	
Less than 30 minutes	4 (3)
30 minutes to 1 hour	16 (13)

Between 1-2 hours	11 (9)
More than 2 to 3 hours	10 (8)
More than 3 to 5 hours	7 (6)
More than 5 hours	0 (--)
Not applicable	71 (60)
What is cooked with primary cook fuel (7 day), n(%)	
Vegetables	118 (99)
Rice	116 (97)
Chiappati	112 (94)
Water	66 (55)
Dal	114 (96)
Meat	103 (87)
What is cooked with secondary cook fuel (7 day), n(%)	
Vegetables	9 (8)
Rice	9 (8)
Chiappati	19 (16)
Water	42 (34)
Dal	10 (8)
Meat	7 (6)
Where is the cook stove used, among those reporting use?, n(%)	
LPG	
In the kitchen/cooking area	101 (100)
Outside the house	0 (--)
Kerosene/paraffin	
In the kitchen/cooking area	26 (93)
Outside the house	2 (7)
Biomass (wood)	
In the kitchen/cooking area	4 (14)
Outside the house	25 (86)
How often put new fuel into cooking fire, among those using wood, n(%)	
Never	8 (22)
Only a few times, so that flame is small and sometimes goes out	7 (19)
Regularly to maintain an obvious flame	22 (59)
How often do you open the windows when cooking?, n(%)	
Never	9 (8)
Less than half of the time	6 (5)
About half of the time	0 (--)
More than half of the time, but not always	11 (9)
Always	55 (46)
Not applicable	38 (32)
How often do you open the doors when cooking?, n(%)	
Never	5 (4)
Less than half of the time	6 (5)
About half of the time	1 (1)
More than half of the time, but not always	23 (19)
Always	82 (69)
Not applicable	2 (2)
How often child near while cooking (7 days), n(%)	
Never	36 (57)

Less than half of the time	17 (27)
About half of the time	1 (2)
More than half of the time, but not always	5 (8)
Always	4 (6)
Time carrying child while cooking (7 days), median (IQR)	30 (30, 60)
Cooking responsibility, n(%)	
All of it	31 (55)
Most of it	8 (14)
Some of it	15 (27)
None	2 (4)
Age started cooking, n(%)	15 (12, 17)

Table 3.2. Kitchen area characteristics and construction materials in low-income urban Indian households (n = 119).

	n=119
Cooking Area, n(%)	
Outside	2 (2)
Inside, separate kitchen	62 (52)
Inside, no separate kitchen	55 (46)
Type of fuel being monitored, n(%)	
Electricity	1 (1)
LPG or Electricity	100 (84)
Paraffin/kerosene	14 (12)
Wood	4 (3)
Volume of cooking area, cubic meters, median (IQR)	405 (28, 734)
Primary construction material of the kitchen walls, n(%)	
Not applicable, outdoor kitchen	0 (--)
Concrete	88 (74)
Brick	6 (5)
Corrugated Metal	25 (21)
Wood	0 (--)
Thatch	0 (--)
Primary construction material of the kitchen roof, n(%)	
Not applicable, outdoor kitchen	1 (1)
Concrete	56 (47)
Brick	0 (--)
Corrugated Metal	53 (45)
Wood	9 (8)
Thatch	0 (--)
Construction material of the kitchen	
All corrugated metal	25 (21)
Roof or walls corrugated metal	38 (32)
All concrete or brick	56 (47)
Doors opening to the outside areas are made of: n(%)	
No doors opening to the outside	7 (6)
Not applicable, hollow/always open	9 (8)
Cloth, bamboo, or other soft material	2 (2)
Wood, glass, or other hard material	100 (84)
Doors opening to the inside areas are made of:, n(%)	
No doors opening to the inside	60 (50)
Not applicable, hollow/always open	46 (39)
Cloth, bamboo, or other soft material	0 (--)
Wood, glass, or other hard material	12 (11)
Windows opening to the outside areas are made of:, n(%)	
No windows opening to the outside	51 (43)
Hollow/always open	22 (18)
Cloth, bamboo, or other soft material	1 (1)
Wood, glass, or other hard material	44 (37)
Is there a visible gap between the roof and the top of the walls?, n(%)	
No	95 (80)

Yes	23 (19)
Size of gap (cm), median (IQR)	4 (3, 6)
What type of road does the house lie on, n(%)	
Small footpath or walking alleyway	74 (62)
Small vehicle alleyway with little traffic	30 (25)
Medium road with moderate traffic	12 (10)
Large thoroughfare with heavy traffic	2 (2)
Are there any large roadways within 100 meters?, n(%)	
No	25 (21)
Yes	93 (78)

Table 3.3. Reported exposure to non-cooking related household air pollutants in low-income urban Indian households (n = 119).

	Total n(%)
How often do you or your neighbors burn trash near your home?, n(%)	
Daily	7 (6)
Weekly	19 (16)
Monthly	6 (5)
Sometimes, but not every month	5 (4)
Never	82 (69)
Burning incense (7 days), n(%)	
No	17 (14)
Yes	102 (86)
Minutes per day burning incense (7 day), median (IQR)	15 (10, 24)
Use mosquito coils (7 day), n(%)	
No	89 (75)
Yes	30 (25)
Hours per day burning mosquito coils (7 day), median (IQR)	450 (60, 600)
Light source (7 days), n(%)	
Kerosene	13 (11)
Candles	82 (69)
Smell others using biomass (7 days), n(%)	
Every day	25 (36)
Not every day, but more than once per week	6 (9)
Once per week	1 (1)
Not every week, but more than once per month	1 (1)
Once per month	2 (3)
Never	34 (49)
Smell others preparing mishri (7 days), n(%)	
Every day	9 (13)
Not every day, but more than once per week	6 (9)
Once per week	18 (26)
Not every week, but more than once per month	8 (12)
Once per month	7 (10)
Never	21 (30)

Table 3.4. Concentrations of markers of household air pollution by reported cooking fuel in low-income urban Indian households (n = 119).

		Primary				**Composite**			
	Total n=119	**LPG n=101**	**Kerosene n=14**	**Wood n=4**	**p-value**	**LPG n=61**	**Kerosene (no wood) n=26**	**Wood n=32**	**p-value**
24-hour mean PM2.5 (μg/m^3), median (IQR)	184 (113, 347)	184 (113, 330)	210 (118, 272)	972 (587, 1274)	0.20	154 (92, 237)	187 (119, 299)	341 (216, 538)	**0.0003**
Hours > 75 μg/m3 PM2.5 (μg/m^3), median (IQR)	11.6 (5.8, 18.8)	11.7 (5.7, 19.0)	11.5 (6.1, 18.3)	11.6 (11.0, 13.5)	0.95	11.1 (4.2, 18.4)	11.3 (6.5, 16.5)	17.1 (9.4, 20.2)	0.09
24-hour mean CO (ppm)†, median (IQR)	3.7 (1.9, 6.3)	**3.5 (1.8, 5.6)**	**5.2 (3.0, 7.1)**	**12.0 (8.2, 18.8)**	**0.02**	**3.0 (1.69, 4.6)**	**5.2 (3.1, 7.9)**	**4.3 (3.4, 6.4)**	**0.03**
Air Nicotine (n=99)									
Detectable									
No	65 (66)	53 (65)	9 (64)	3 (75)	1.0	29 (63)	16 (64)	20 (71)	0.80
Yes	34 (34)	28 (35)	5 (36)	1 (25)		17 (37)	9 (36)	8 (29)	
Categorical									
Undetectable	65 (69)	53 (68)	9 (75)	3 (75)	0.39	29 (66)	16 (70)	20 (74)	0.69
Low Detectable	14 (15)	14 (18)	0 (--)	0 (--)		9 (20)	2 (9)	3 (11)	
High Detectable	15 (16)	11 (14)	3 (25)	1 (25)		6 (14)	5 (22)	4 (15)	
Air nicotine among detectable (μg/m^3), median (IQR)	0.093 (0.014, 0.22)	0.078 (0.010, 0.21)	0.19 (0.093, 0.25)	0.10 (0.10, 0.10)	0.25	0.078 (0.01, 0.15)	0.19 (0.093, 0.23)	0.10 (0.01, 0.30)	0.38

† n=117 observations for CO measurements

Figure 3.1. Mean 24-hour average $PM_{2.5}$ concentration ($\mu g/m^3$) by type of a) primary cooking fuel and b) composite cooking fuel in low-income urban Indian households (n = 119).

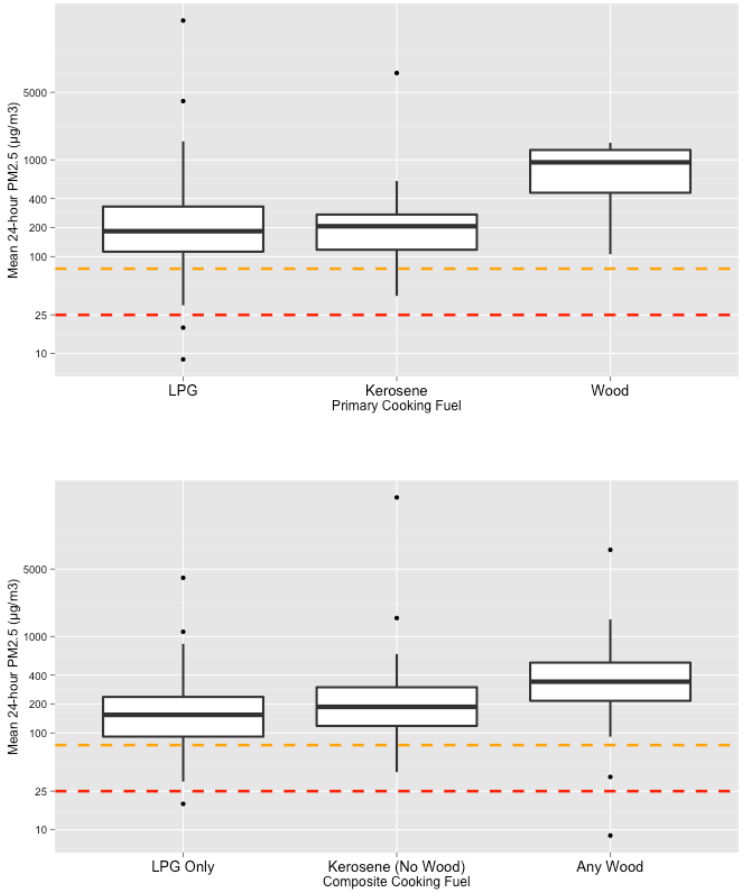


Figure 3.2. Number of hours of $PM_{2.5}$ above 75 $\mu g/m^3$ by type of a) primary cooking fuel and b) secondary cooking fuel in low-income urban Indian households (n = 119).

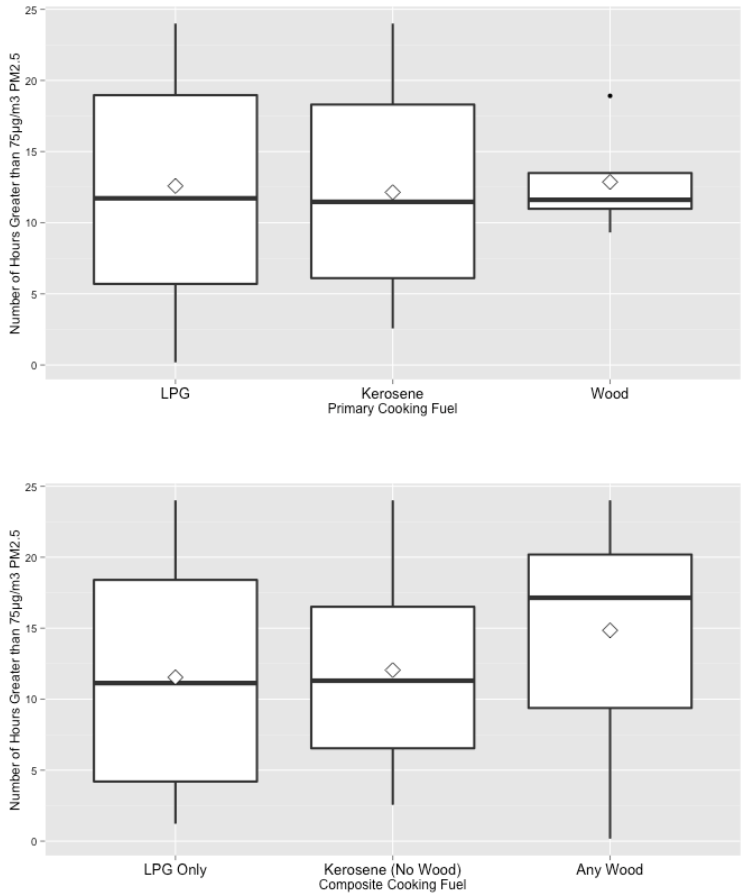


Figure 3.3. Mean 24-hour average CO concentration (ppm) by type of a) primary cooking fuel and b) composite cooking fuel in low-income urban Indian households (n = 117).

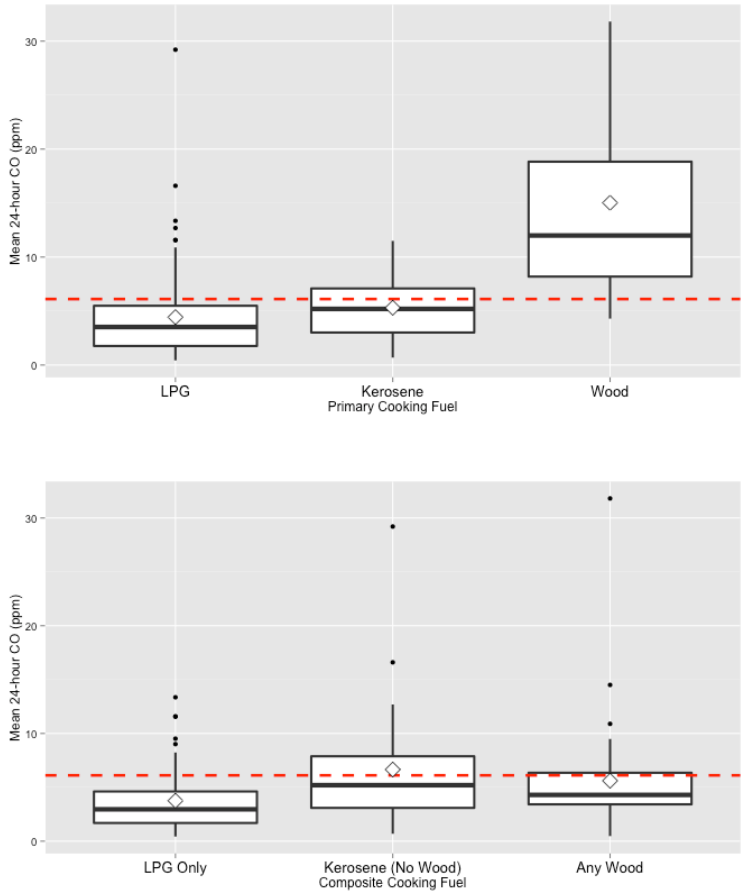


Figure 3.4. Median of the 15 minute moving average of $PM_{2.5}$ ($\mu g/m^3$) by a) primary cooking fuel and b) composite coking fuel in low-income urban Indian households (n = 119).

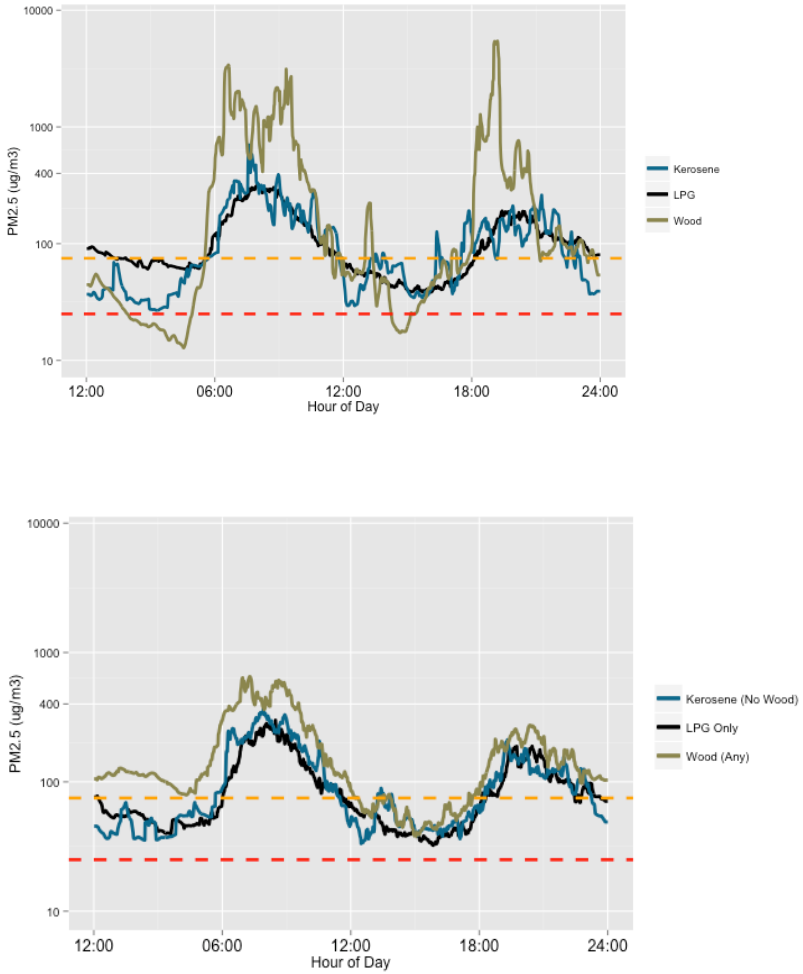


Figure 3.5. Median of the 15 minute moving average of CO (ppm) by a) primary cooking fuel and b) composite cooking fuel in low-income urban Indian households (n = 119).

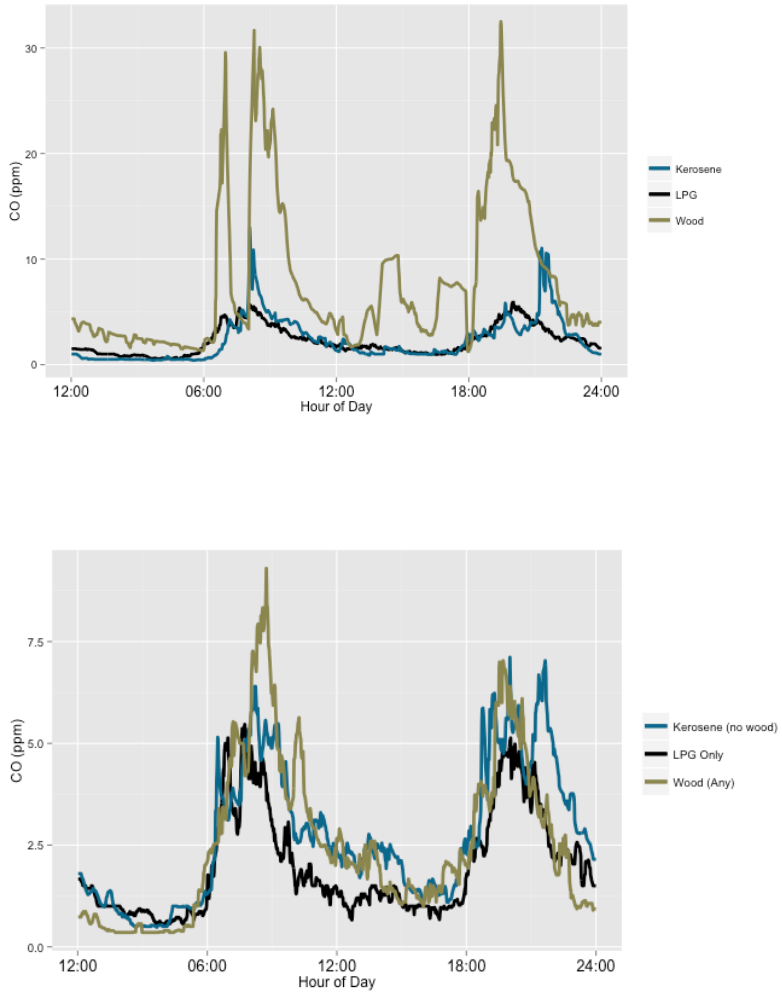


Figure 3.6. Minute by minute median of the moving 15 minute average $PM_{2.5}$ ($\mu g/m^3$), by quartile of 24-hour average $PM_{2.5}$ concentration among 119 low-income urban Indian households.

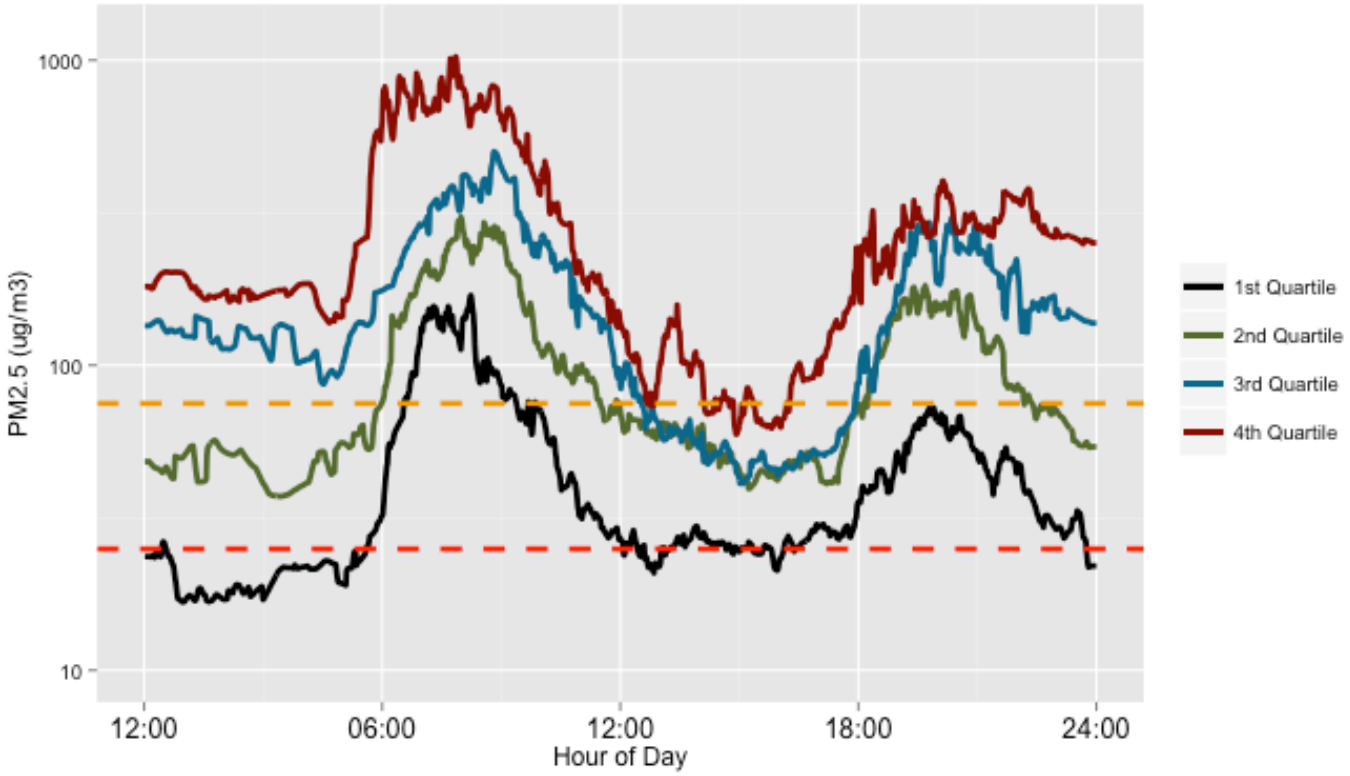


Table 3.5. Univariate linear regression for continuous log 24-hour average $PM_{2.5}$ ($\mu g/m^3$) and continuous log 24-hour average CO (ppm) concentrations across reported measures of exposure to cooking fuel, cooking fuel behaviors, and other sources of HAP among households in low-income urban Indian households (n = 119).

	Log 24-hour average $PM_{2.5}$ ($\mu g/m^3$)		**Log 24-hour average CO (ppm)**	
	Univariate Coef. (95% CI)	**p-value***	**Univariate Coef. (95% CI)**	**p-value***
Primary Cooking Fuel				
LPG	REF		REF	
Kerosene	0.10 (-0.52, 0.71)	0.76	0.28 (-0.28, 0.79)	0.28
Composite Fuel Types				
LPG/electricity only	REF		REF	
Kerosene (no biomass)	0.40 (-0.09, 0.90)	0.11	**0.49 (0.08, 0.90)**	**0.02**
Any biomass	**0.74 (0.28, 1.20)**	**0.002**	0.33 (-0.06, 0.72)	0.09
Composite Fuel Types – no wood†				
LPG/electricity only	REF		REF	
Kerosene (no biomass)	0.40 (-0.09, 0.89)	0.11	**0.49 (0.08, 0.89)**	**0.02**
Any biomass	**0.65 (0.17, 1.13)**	**0.01**	0.17 (-0.23, 0.56)	0.41
Construction material of the kitchen				
All corrugated metal	REF		REF	
Roof or walls corrugated metal	-0.49 (-1.02, 0.04)	0.07	0.01 (-0.45, 0.48)	0.95
All concrete or brick	**-0.98 (-1.48, -0.49)**	**0.0001**	-0.12 (-0.56, 0.31)	0.57
Rudimentary Floor Material				
No	REF		REF	
Yes	-0.21 (-1.50, 1.07)	0.742	0.99 (-0.27, 2.25)	0.12
Volume of Cooking Area Volume				
≤ Median	REF		REF	
> Median	-0.24 (-0.64, 0.16)	0.24	-0.21 (-0.54, 0.12)	0.20
Always opening door				
No				
Yes	-0.02 (-0.46, 0.42)	0.93	-0.01 (-0.37, 0.35)	0.97
Always opening window				
No				

Yes	-0.39 (-0.78, 0.001)	0.05	-0.10 (-0.43, 0.24)	0.57
Detectable air nicotine				
No	REF		REF	
Yes	-0.18 (-0.64, 0.28)	0.43	-0.07 (-0.44, 0.31)	0.72
Categorical air nicotine				
Undetectable	REF		REF	
Low detectable	-0.52 (-1.17, 0.14)	0.12	-0.10 (-0.61, 0.41)	0.70
High detectable	0.13 (-0.50, 0.77)	0.68	0.13 (-0.37, 0.63)	0.60
Visible gap between wall and ceiling				
No	REF		REF	
Yes	**0.80 (0.31, 1.29)**	**0.002**	0.21 (-0.20, 0.62)	0.32
Smell others using biomass daily				
No	REF		REF	
Yes	0.17 (-0.40, 0.74)	0.55	0.21 (-0.24, 0.65)	0.36
Smell others using mishri daily				
No	REF		REF	
Yes	-0.03 (-0.60, 0.54)	0.92	-0.06 (-0.51, 0.38)	0.79
Use of primary cook fuel 3+ hours per day				
No	REF		REF	
Yes	-0.003 (-0.40, 0.40)	0.99	0.03 (-0.30, 0.36)	0.88
What type of road does the house lie on				
Small footpath or walking alleyway	REF		REF	
Small vehicle alleyway with little traffic	-0.37 (-0.84, 0.10)	0.13	0.06 (-0.32, 0.44)	0.75
Medium road with moderate traffic	-0.001 (-0.68, 0.68)	1.0	0.09 (-0.45, 0.63)	0.75
Large thoroughfare with heavy traffic	-1.09 (-2.66, 0.48)	0.17	-0.83 (-2.08, 0.43)	0.19
Large roadways within 100 meters				
No	REF		REF	
Yes	0.02 (-0.47, 0.52)	0.93	0.04 (-0.36, 0.44)	0.84
Burn trash at least weekly				
No	REF		REF	
Yes	-0.05 (-0.54, 0.44)	0.84	-0.09 (-0.50, 0.31)	0.64
Burning incense				

No	REF			
Yes	**-0.59 (-1.15, -0.03)**	**0.04**	-0.29 (-0.77, 0.18)	0.23
Use mosquito coils				
No	REF		REF	
Yes	0.24 (-0.22, 0.70)	0.31	-0.19 (-0.57, 0.19)	0.32
Light source - Kerosene				
No	REF		REF	
Yes	0.26 (-0.38, 0.91)	0.42	0.30 (-0.23, 0.82)	0.26
Light Source – Candles				
No	REF		REF	
Yes	0.06 (-0.37, 0.50)	0.77	-0.14 (-0.49, 0.23)	0.44
Winter season				

*Bolded values are statistically significant at $p < 0.05$

†Restricted data set excluding households reporting wood as primary cooking fuel

Table 3.6. Multivariate linear regression for continuous log 24-hour average $PM_{2.5}$ ($\mu g/m^3$) concentration controlling for reported measures of exposure to cooking fuel, cooking fuel behaviors, and other sources of HAP among households in low-income urban Indian households (n = 119).

	Univariate Coef. (95% CI)	**p-value***	**Multivariate Coef. (95% CI)**	**p-value***
Primary Cooking Fuel†				
LPG/electricity only	REF		REF	
Kerosene	0.10 (-0.52, 0.71)	0.76	-0.07 (-0.70, 0.55)	0.82
Construction material of the kitchen				
All corrugated metal	REF		REF	
Roof or walls corrugated metal	-0.49 (-1.02, 0.04)	0.07	-0.32 (-0.98, 0.34)	0.34
All concrete or brick	**-0.98 (-1.48, -0.49)**	**0.0001**	**-0.82 (-1.48, -0.16)**	**0.02**
Volume of Cooking Area > Median	-0.24 (-0.64, 0.16)	0.24	-0.10 (-0.51, 0.32)	0.64
Always open window when cooking	-0.39 (-0.78, 0.001)	0.05	-0.17 (-0.64, 0.31)	0.49
Always open door when cooking	-0.02 (-0.46, 0.42)	0.93	-0.09 (-0.57, 0.40)	0.72
Uses mosquito coils	0.24 (-0.22, 0.70)	0.31	0.37 (-0.07, 0.81)	0.10
Winter Season	0.35 (-0.06, 0.76)	0.09	**0.47 (0.05, 0.89)**	**0.03**
Composite Cooking Fuel				
LPG/electricity only	REF		REF	
Kerosene (no biomass)	0.40 (-0.09, 0.90)	0.11	0.26 (-0.25, 0.77)	0.32
Any wood	**0.74 (0.28, 1.20)**	**0.002**	0.38 (-0.14, 0,90)	0.15
Construction material of the kitchen				
All corrugated metal	REF		REF	
Roof or walls corrugated metal	-0.49 (-1.02, 0.04)	0.07	-0.31 (-0.97, 0.36)	0.36
All concrete or brick	**-0.98 (-1.48, -0.49)**	**0.0001**	**-0.81 (-1.49, -0.13)**	**0.02**
Volume of Cooking Area > Median	-0.24 (-0.64, 0.16)	0.24	0.004 (-0.40, 0.41)	0.98
Always open window when cooking	-0.39 (-0.78, 0.001)	0.05	-0.13 (-0.60, 0.34)	0.58
Always open door when cooking	-0.02 (-0.46, 0.42)	0.93	-0.11 (-0.59, 0.36)	0.63
Uses mosquito coils	0.24 (-0.22, 0.70)	0.31	0.25 (-0.19, 0.70)	0.27
Winter Season	0.35 (-0.06, 0.76)	0.09	**0.45 (0.03, 0.87)**	**0.04**

Composite Cooking Fuel† – no primary wood				
LPG/electricity only	REF		REF	
Kerosene (no biomass)	0.40 (-0.09, 0.89)	0.11	0.31 (-0.20, 0.82)	0.23
Any wood	**0.65 (0.17, 1.13)**	**0.008**	0.35 (-0.19, 0.88)	0.20
Construction material of the kitchen				
All corrugated metal	REF		REF	
Roof or walls corrugated metal	-0.49 (-1.02, 0.04)	0.07	-0.10 (-0.78, 0.59)	0.77
All concrete or brick	**-0.98 (-1.48, -0.49)**	**0.0001**	-0.56 (-1.26, 0.15)	0.12
Volume of Cooking Area > Median	-0.24 (-0.64, 0.16)	0.24	-0.08 (-0.48, 0.33)	0.71
Always open window when cooking	-0.39 (-0.78, 0.001)	0.05	-0.22 (-0.70, 0.26)	0.36
Always open door when cooking	-0.02 (-0.46, 0.42)	0.93	-0.01 (-0.50 0.47)	0.95
Use mosquito coils	0.24 (-0.22, 0.70)	0.31	0.30 (-0.14, 0.75)	0.18
Winter Season	0.35 (-0.06, 0.76)	0.09	**0.49 (0.07, 0.91)**	**0.02**

*Bolded values are statistically significant at $p < 0.05$

†Restricted data set excluding households reporting wood as primary cooking fuel

Table 3.7. Multivariate linear regression for continuous log 24-hour average CO (ppm) concentration controlling for reported measures of exposure to cooking fuel, cooking fuel behaviors, and other sources of HAP among households in low-income urban Indian households (n = 117).

	Univariate Coef. (95% CI)	**p-value***	**Multivariate Coef. (95% CI)**	**p-value***
Primary Cooking Fuel†				
LPG/electricity only	REF		REF	
Kerosene	0.28 (-0.28, 0.79)	0.28	0.48 (-0.06, 1.01)	0.08
Construction material of the kitchen				
All corrugated metal	REF		REF	
Roof or walls corrugated metal	0.01 (-0.45, 0.48)	0.95	0.39 (-0.16, 0.94)	0.16
All concrete or brick	-0.12 (-0.56, 0.31)	0.57	0.29 (-0.26, 0.85)	0.31
Volume of Cooking Area > Median	-0.21 (-0.54, 0.12)	0.20	-0.20 (-0.55, 0.14)	0.25
Always open window when cooking	-0.10 (-0.43, 0.24)	0.57	-0.20 (-0.60, 0.20)	0.33
Always open door when cooking	-0.01 (-0.37, 0.35)	0.97	0.20 (-0.21, 0.61)	0.34
Uses mosquito coils	-0.19 (-0.57, 0.19)	0.32	-0.07 (-0.44, 0.30)	0.71
Winter Season	**0.39 (0.05, 0.72)**	**0.02**	**0.38 (0.03, 0.73)**	**0.03**
Composite Cooking Fuel				
LPG/electricity only	REF		REF	
Kerosene (no biomass)	**0.49 (0.08, 0.90)**	**0.02**	**0.66 (0.23, 1.10)**	**0.003**
Any wood	0.33 (-0.06, 0.72)	0.09	0.44 (-0.01, 0.88)	0.055
Construction material of the kitchen				
All corrugated metal	REF		REF	
Roof or walls corrugated metal	0.01 (-0.45, 0.48)	0.95	0.27 (-0.29, 0.84)	0.34
All concrete or brick	-0.12 (-0.56, 0.31)	0.57	0.16 (-0.42, 0.74)	0.58
Volume of Cooking Area > Median	-0.21 (-0.54, 0.12)	0.20	-0.02 (-0.36, 0.33)	0.92
Always open window when cooking	-0.10 (-0.43, 0.24)	0.57	-0.13 (-0.53. 0.23)	0.54
Always open door when cooking	-0.01 (-0.37, 0.35)	0.97	0.11 (-0.29, 0.52)	0.58
Uses mosquito coils	-0.19 (-0.57, 0.19)	0.32	-0.27 (-0.65, 0.11)	0.16
Winter Season	**0.39 (0.05, 0.72)**	**0.02**	**0.42 (0.06, 0.77)**	**0.02**

Composite Cooking Fuel† – no wood as primary				
LPG/electricity only	REF		REF	
Kerosene (no biomass)	**0.49 (0.08, 0.89)**	**0.02**	**0.71 (0.22, 1.07)**	**0.001**
Any wood	0.17 (-0.23, 0.56)	0.41	0.32 (-0.13, 0.77)	0.15
Construction material of the kitchen				
All corrugated metal	REF		REF	
Roof or walls corrugated metal	0.01 (-0.45, 0.48)	0.95	0.53 (0.01, 0.15)	0.06
All concrete or brick	-0.12 (-0.56, 0.31)	0.57	0.44 (-0.09, 1.10)	0.14
Volume of Cooking Area > Median	-0.21 (-0.54, 0.12)	0.20	-0.10 (-0.56, 0.10)	0.55
Always open window when cooking	-0.10 (-0.43, 0.24)	0.57	-0.27 (-0.69, 0.11)	0.18
Always open door when cooking	-0.01 (-0.37, 0.35)	0.97	-0.24 (-0.23, 0.58)	0.23
Use mosquito coils	-0.19 (-0.57, 0.19)	0.32	-0.18 (-0.58, 0.17)	0.33
Winter Season	**0.39 (0.05, 0.72)**	**0.02**	**0.45 (0.10, 0.79)**	**0.01**

*Bolded values are statistically significant at $p < 0.05$

†Restricted data set excluding households reporting wood as primary cooking fuel

Table 3.8. Univariate logistic regression for a) odds of having a larger number of hours with a $PM_{2.5}$ above 75 $\mu g/m^3$ greater than the median for the group and b) for being categorized as second through fourth quartile of average 24-hour $PM_{2.5}$ as compared to the first quartile, by reported measures of exposure to cooking fuel, cooking fuel behaviors, and other sources of HAP among households in low-income urban Indian households (n = 119).

	a) Hrs > 75		**b) 2nd-4th Quartile vs 1st Quartile**	
	Univariate OR (95% CI)	**p-value**	**Univariate OR (95% CI)**	**p-value**
Primary Cooking Fuel				
LPG	REF		REF	
Kerosene	0.52 (0.15, 1.62)	0.27	1.27 (0.6, 5.94)	0.73
Composite Fuel Types†, n(%)				
LPG/electricity only	REF		REF	
Kerosene (no biomass)	0.69 (0.26, 1.74)	0.44	3.10 (1.03, 11.63)	0.06
Any biomass	1.84 (0.77, 4.50)	0.17	**3.95 (1.33, 14.63)**	**0.02**
Construction material of the kitchen				
All corrugated metal	REF		REF	
Roof or walls corrugated metal	0.77 (0.27, 2.17)	0.63	0.33 (0.05, 1.45)	0.18
All concrete or brick	**0.34 (0.12, 0.88)**	**0.03**	0.16 (0.02, 0.61)	0.02
Rudimentary Floor Material	--	--	--	--
Cooking area volume (binary above median)	**0.37 (0.17, 0.77)**	**0.008**	0.47 (0.20, 1.09)	0.08
Always opening door	0.77 (0.34, 1.69)	0.51	0.84 (0.32, 2.05)	0.70
Always opening window	0.56 (0.27, 1.16)	0.12	**0.43 (0.18, 1.00)**	**0.05**
Detectable air nicotine				
No	REF		REF	
Yes	0.81 (0.35, 1.85)	0.61	1.26 (0.47, 3.62)	0.65
Categorical air nicotine				
Undetectable	REF		REF	
Low detectable	0.60 (0.18, 1.93)	0.40	0.59 (0.18, 2.14)	0.40
High detectable	2.22 (0.68, 8.66)	0.21	4.57 (0.82, 86.05)	0.16
Visible gap between wall and ceiling	1.80 (0.72, 4.71)	0.21	2.65 (0.82, 11.89)	0.14
Smell others using biomass daily	1.29 (0.50, 3.39)	0.60	1.38 (0.50, 3.95)	0.53
Smell others using mishri daily	0.57 (0.21, 1.49)	0.26	0.88 (0.32, 2.44)	0.80

Use of primary cook fuel 3+ hours per day, n(%)	0.69 (0.33, 1.42)	0.31	0.71 (0.31, 1.64)	0.43
What type of road does the house lie on, n(%)				
Small footpath or walking alleyway	--	--	--	--
Small vehicle alleyway with little traffic	--	--	--	--
Medium road with moderate traffic	--	--	--	--
Large thoroughfare with heavy traffic	--	--	--	--
Are there any large roadways within 100 meters?, n(%)				
No	REF		REF	
Yes	0.87 (0.35, 2.10)	0.75	1.93 (0.73, 4.94)	0.18
Burn trash at least weekly?, n(%)	01.86 (0.77, 4.65)	0.17	0.89 (0.34, 2.52)	0.82
Burning incense (7 days), n(%)				
No	REF		REF	
Yes	0.25 (0.07, 0.77)	0.23	0.16 (0.01, 0.83)	0.08
Use mosquito coils (7 day), n(%)				
No	REF		REF	
Yes	1.02 (0.44, 2.35)	0.96	1.48 (0.56, 4.38)	0.45
Light source (7 days), n(%)				
Kerosene	0.41 (0.11, 1.35)	0.16	0.73 (0.22, 2.88)	0.63
Candles	0.90 (0.41, 1.97)	0.80	0.93 (0.37, 2.25)	0.88

*Bolded values are statistically significant at $p < 0.05$

†Restricted data set excluding households reporting wood as primary cooking fuel

Table 3.9. Multivariate logistic regression for odds of having a larger number of hours with a $PM_{2.5}$ above 75 $\mu g/m^3$ greater than the median for the group controlling for reported measures of exposure to cooking fuel, cooking fuel behaviors, and other sources of HAP among households in low-income urban Indian households (n = 119).

	Univariate OR (95% CI)	**p-value**	**Adjusted OR (95% CI)**	**p-value**
Primary Fuel Types, n(%)				
LPG/electricity only	REF		REF	
Kerosene	0.52 (0.15, 1.62)	0.27	0.28 (0.06, 1.26)	0.11
Construction material of the kitchen				
All corrugated metal	REF		REF	
Roof or walls corrugated metal	0.77 (0.27, 2.17)	0.63	0.39 (0.08, 1.70)	0.22
All concrete or brick	**0.34 (0.12, 0.88)**	**0.03**	**0.12 (0.02, 0.57)**	**0.01**
Binary Cooking Volume (greater than median)	**0.37 (0.17, 0.77)**	**0.008**	**0.64 (0.26, 1.61)**	**0.34**
Always open window when cooking	0.56 (0.27, 1.16)	0.12	0.82 (0.27, 2.44)	0.71
Always open door when cooking	0.77 (0.34, 1.69)	0.51	0.55 (0.18, 1.66)	0.29
Use mosquito coils	1.02 (0.44, 2.35)	0.96	1.61 (0.57, 4.73)	037
Winter season	**6.8 (2.99, 16.23)**	**<0.0001**	**10.11 (3.74, 30.84)**	**<0.0001**
Composite Fuel Types, n(%)				
LPG/electricity only	REF		REF	
Kerosene (no biomass)	0.69 (0.26, 1.74)	0.44	0.49 (0.14, 1.59)	0.24
Any wood	1.84 (0.77, 4.50)	0.17	0.98 (0.28, 3.36)	0.97
Construction material of the kitchen				
All corrugated metal	REF		REF	
Roof or walls corrugated metal	0.77 (0.27, 2.17)	0.63	0.47 (0.10, 2.09)	0.32
All concrete or brick	**0.34 (0.12, 0.88)**	**0.03**	**0.15 (0.03, 0.69)**	**0.02**
Binary Cooking Volume (greater than median)	**0.37 (0.17, 0.77)**	**0.008**	0.61 (0.25, 1.49)	0.28
Always open window when cooking	0.56 (0.27, 1.16)	0.12	0.85 (0.30, 2.47)	0.77
Always open door when cooking	0.77 (0.34, 1.69)	0.51	0.65 (0.22, 1.87)	0.43
Use mosquito coils	1.02 (0.44, 2.35)	0.96	1.70 (0.59, 5.06)	0.33
Winter season	**6.8 (2.99, 16.23)**	**<0.0001**	**8.70 (3.27, 25.92)**	**<0.0001**

Composite Fuel Types†, n(%) – no wood as primary				
LPG/electricity only	REF		REF	
Kerosene (no biomass)	0.69 (0.26, 1.74)	0.44	0.52 (0.15, 1.71)	0.29
Any wood	1.99 (0.80, 5.13)	0.15	1.15 (0.31, 4.21)	0.83
Construction material of the kitchen				
All corrugated metal	REF		REF	
Roof or walls corrugated metal	0.77 (0.27, 2.17)	0.63	0.55 (0.11, 2.63)	0.45
All concrete or brick	**0.34 (0.12, 0.88)**	**0.03**	**0.18 (0.03, 0.94)**	**0.05**
Binary Cooking Volume (greater than median)	**0.37 (0.17, 0.77)**	**0.008**	0.55 (0.22, 1.39)	0.21
Always open window when cooking	0.56 (0.27, 1.16)	0.12	0.82 (0.27, 2.51)	0.73
Always open door when cooking	0.77 (0.34, 1.69)	0.51	0.60 (0.20, 1.81)	0.37
Use mosquito coils	1.02 (0.44, 2.35)	0.96	1.66 (0.57, 5.04)	0.36
Winter season	**6.8 (2.99, 16.23)**	**<0.0001**	**9.25 (3.43, 27.95)**	**<0.0001**

*Bolded values are statistically significant at $p < 0.05$

†Restricted data set excluding households reporting wood as primary cooking fuel

Table 3.10. Multivariate logistic regression for odds of being categorized as second through fourth quartile of average 24-hour $PM_{2.5}$ as compared to the first quartile controlling for reported measures of exposure to cooking fuel, cooking fuel behaviors, and other sources of HAP among households in low-income urban Indian households (n = 119).

	Univariate OR (95% CI)	**p-value**	**Adjusted OR (95% CI)**	**p-value**
Primary Fuel Types, n(%)				
LPG/electricity only	REF		REF	
Kerosene	1.27 (0.6, 5.94)	0.73	1.05 (0.22, 5.88)	0.96
Construction material of the kitchen				
All corrugated metal	REF		REF	
Roof or walls corrugated metal	0.33 (0.05, 1.45)	0.18	0.39 (0.05, 2.35)	0.33
All concrete or brick	**0.16 (0.02, 0.61)**	**0.02**	0.21 (0.03, 1.20)	0.10
Binary Cooking Volume (greater than median)	0.47 (0.20, 1.09)	0.08	0.56 (0.21, 1.50)	0.25
Always open window when cooking	**0.43 (0.18, 1.00)**	**0.05**	0.64 (0.21, 1.89)	0.42
Always open door when cooking	0.84 (0.32, 2.05)	0.70	0.96 (0.30, 3.03)	0.95
Use mosquito coils	1.48 (0.56, 4.38)	0.45	2.05 (0.70, 6.76)	0.21
Winter season	2.34 (0.95, 6.41)	0.08	**3.00 (1.04, 9.66)**	**0.05**
Composite Fuel Types, n(%)				
LPG/electricity only	REF		REF	
Kerosene (no biomass)	3.10 (1.03, 11.63)	0.06	2.75 (0.79, 11.44)	0.13
Any wood	**3.95 (1.33, 14.63)**	**0.02**	2.54 (0.67, 1.40)	0.19
Construction material of the kitchen				
All corrugated metal	REF		REF	
Roof or walls corrugated metal	0.33 (0.05, 1.45)	0.18	0.61 (0.07, 4.10)	0.62
All concrete or brick	0.16 (0.02, 0.61)	0.02	0.30 (0.03, 1.89)	0.23
Binary Cooking Volume (greater than median)	0.47 (0.20, 1.09)	0.08	0.70 (0.26, 1.89)	0.48
Always open window when cooking	**0.43 (0.18, 1.00)**	**0.05**	0.53 (0.17, 1.54)	0.25
Always open door when cooking	0.84 (0.32, 2.05)	0.70	0.96 (0.30, 2.92)	0.94
Use mosquito coils	1.48 (0.56, 4.38)	0.45	1.76 (0.57, 6.03)	0.34
Winter season	2.34 (0.95, 6.41)	0.08	2.75 (0.97, 8.58)	0.07

Composite Fuel Types†, n(%) – no wood as primary				
LPG/electricity only	REF		REF	
Kerosene (no biomass)	3.10 (1.03, 11.63)	0.06	3.01 (0.86, 12.66)	0.10
Any wood	**4.70 (1.44, 21.33)**	**0.02**	3.58 (0.87, 19.07)	0.10
Construction material of the kitchen				
All corrugated metal	REF		REF	
Roof or walls corrugated metal	0.33 (0.05, 1.45)	0.18	0.85 (0.09, 6.06)	0.87
All concrete or brick	0.16 (0.02, 0.61)	0.02	0.50 (0.06, 3.33)	0.50
Binary Cooking Volume (greater than median)	0.47 (0.20, 1.09)	0.08	0.61 (0.22, 1.67)	0.34
Always open window when cooking	**0.43 (0.18, 1.00)**	**0.05**	0.51 (0.16, 1.55)	0.24
Always open door when cooking	0.84 (0.32, 2.05)	0.70	1.13 (0.35, 3.53)	0.84
Use mosquito coils	1.48 (0.56, 4.38)	0.45	1.74 (0.55, 6.07)	0.36
Winter season	2.34 (0.95, 6.41)	0.08	**3.31 (1.12, 10.99)**	**0.04**

*Bolded values are statistically significant at $p < 0.05$

†Restricted data set excluding households reporting wood as primary cooking fuel

Table 3.11. Univariate logistic regression for odds of being categorized as fourth quartile of average 24-hour $PM_{2.5}$ as compared to the first through the third quartile, by reported measures of exposure to cooking fuel, cooking fuel behaviors, and other sources of HAP among households in low-income urban Indian households (n = 119).

	Univariate OR (95% CI)	**p-value**
Primary Cooking Fuel		
LPG	REF	
Kerosene	0.88 (0.19, 3.08)	0.85
Composite Fuel Types†, n(%)		
LPG/electricity only	REF	
Kerosene (no biomass)	1.99 (0.59, 6.44)	0.25
Any biomass	**6.63 (2.47, 19.15)**	**0.0001**
Construction material of the kitchen		
All corrugated metal	REF	
Roof or walls corrugated metal	0.52 (0.18, 1.49)	0.22
All concrete or brick	**0.21 (0.07, 0.62)**	**0.005**
Rudimentary Floor Material	--	--
Cooking area volume (binary above median)	1.03 (0.44, 2.35)	0.96
Always opening door	0.96 (0.40, 2.47)	0.93
Always opening window	0.65 (0.28, 1.50)	0.32
Detectable air nicotine		
No	REF	
Yes	**0.26 (0.07, 0.78)**	**0.02**
Categorical air nicotine		
Undetectable	REF	
Low detectable	0.14 (0.01, 0.75)	0.06
High detectable	0.52 (0.11, 1.88)	0.35
Visible gap between wall and ceiling	**3.67 (1.40, 9.69)**	**0.01**
Smell others using biomass daily	2.13 (0.63, 7.84)	0.23
Smell others using mishri daily	0.92 (0.27, 3.11)	0.89
Use of primary cook fuel 3+ hours per day, n(%)	0.82 (0.35, 1.88)	0.63

What type of road does the house lie on, n(%)		
Small footpath or walking alleyway	--	--
Small vehicle alleyway with little traffic	--	--
Medium road with moderate traffic	--	--
Large thoroughfare with heavy traffic	--	--
Are there any large roadways within 100 meters?, n(%)		
No	REF	
Yes	0.52 (0.20, 1.38)	0.18
Burn trash at least weekly?, n(%)	1.12 (0.40, 2.92)	0.82
Burning incense (7 days), n(%)		
No	REF	
Yes	1.11 (0.36, 4.22)	0.86
Use mosquito coils (7 day), n(%)		
No	REF	
Yes	**2.63 (1.06, 6.45)**	**0.03**
Light source (7 days), n(%)		
Kerosene	1.37 (0.35, 4.59)	0.63
Candles	1.67 (0.67, 4.61)	0.29

*Bolded values are statistically significant at $p < 0.05$

†Restricted data set excluding households reporting wood as primary cooking fuel

Table 3.12. Multivariate logistic regression for odds of being categorized as fourth quartile of average 24-hour $PM_{2.5}$ as compared to the first through the third quartile controlling for reported measures of exposure to cooking fuel, cooking fuel behaviors, and other sources of HAP among households in low-income urban Indian households (n = 119).

	Univariate OR (95% CI)	**p-value**	**Adjusted OR (95% CI)**	**p-value**
Primary Fuel Types, n(%)				
LPG/electricity only	REF		REF	
Kerosene	0.88 (0.19, 3.08)	0.85	0.53 (0.09, 2.40	0.43
Construction material of the kitchen				
All corrugated metal	REF		REF	
Roof or walls corrugated metal	0.52 (0.18, 1.49)	0.22	0.53 (0.11, 2.51)	0.42
All concrete or brick	**0.21 (0.07, 0.62)**	**0.005**	**0.16 (0.03, 0.85)**	**0.04**
Binary Cooking Volume (greater than median)	1.03 (0.44, 2.35)	0.96	1.48 (0.52, 4.32)	0.46
Always open window when cooking	0.65 (0.28, 1.50)	0.32	0.85 (0.24, 3.06)	0.80
Always open door when cooking	0.96 (0.40, 2.47)	0.93	0.70 (0.20, 2.51)	0.58
Use mosquito coils	**2.63 (1.06, 6.45)**	**0.03**	**4.37 (1.53, 13.35)**	**0.07**
Winter season	2.07 (0.89, 4.84)	0.09	**4.24 (1.50, 13.09)**	**0.008**
Composite Fuel Types, n(%)				
LPG/electricity only	REF		REF	
Kerosene (no biomass)	1.99 (0.59, 6.44)	0.25	1.86 (0.48, 7.05)	0.36
Any wood	**6.63 (2.47, 19.15)**	**0.0001**	**5.53 (1.62, 20.39)**	**0.007**
Construction material of the kitchen				
All corrugated metal	REF		REF	
Roof or walls corrugated metal	0.52 (0.18, 1.49)	0.22	0.76 (0.15, 3.63)	0.73
All concrete or brick	**0.21 (0.07, 0.62)**	**0.005**	0.24 (0.04, 1.27)	0.10
Binary Cooking Volume (greater than median)	1.03 (0.44, 2.35)	0.96	2.19 (0.77, 6.66)	0.15
Always open window when cooking	0.65 (0.28, 1.50)	0.32	1.02 (0.28, 3.83)	0.98
Always open door when cooking	0.96 (0.40, 2.47)	0.93	0.73 (0.21, 2.66)	0.63
Use mosquito coils	**2.63 (1.06, 6.45)**	**0.03**	**3.41 (1.19, 10.31)**	**0.02**
Winter season	2.07 (0.89, 4.84)	0.09	**4.26 (1.42, 13.95)**	**0.01**

Composite Fuel Types†, n(%) – no wood as primary				
LPG/electricity only	REF		REF	
Kerosene (no biomass)	1.99 (0.59, 6.45)	0.25	2.03 (0.52, 7.78)	0.30
Any wood	**5.74 (2.05, 17.10)**	**0.001**	**5.22 (1.46, 20.05)**	**0.01**
Construction material of the kitchen				
All corrugated metal	REF		REF	
Roof or walls corrugated metal	0.52 (0.18, 1.49)	0.22	1.27 (0.24, 6.91)	0.78
All concrete or brick	**0.21 (0.07, 0.62)**	**0.005**	0.46 (0.07, 2.86)	0.40
Binary Cooking Volume (greater than median)	1.03 (0.44, 2.35)	0.96	1.71 (0.58, 5.27)	0.33
Always open window when cooking	0.65 (0.28, 1.50)	0.32	0.78 (0.20, 3.07)	0.72
Always open door when cooking	0.96 (0.40, 2.47)	0.93	0.94 (0.25, 3.82)	0.93
Use mosquito coils	**2.63 (1.06, 6.45)**	**0.03**	**3.82 (1.31, 11.79)**	**0.02**
Winter season	2.07 (0.89, 4.84)	0.09	**4.47 (1.56, 15.38)**	**0.008**

*Bolded values are statistically significant at $p < 0.05$

†Restricted data set excluding households reporting wood as primary cooking fuel

Figure 3.7. Correlation between log-transformed mean 24-hour $PM_{2.5}$ concentration ($\mu g/m^3$) and mean 24-hour CO concentration (ppm) among 117 low-income urban Indian households.

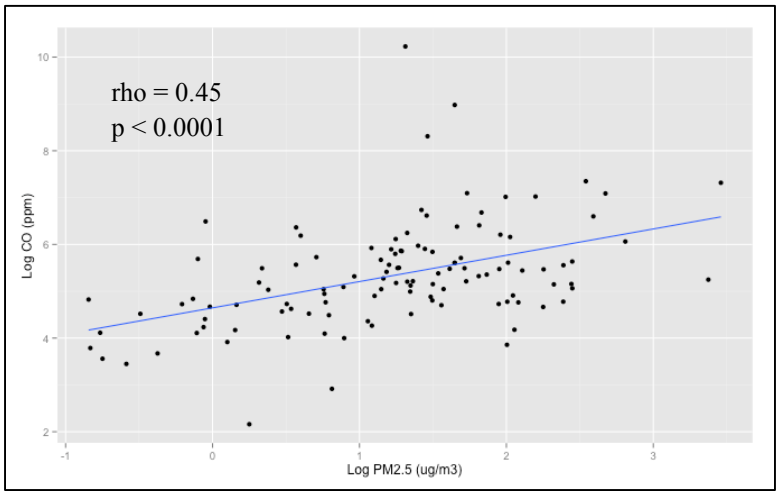


Figure 3.8. Correlation between log-transformed mean 24-hour $PM_{2.5}$ concentration ($\mu g/m^3$) and mean 24-hour CO concentration (ppm) among 117 low-income urban Indian households by primary cooking fuel (a) LPG, b) Kerosene, and c) Wood)

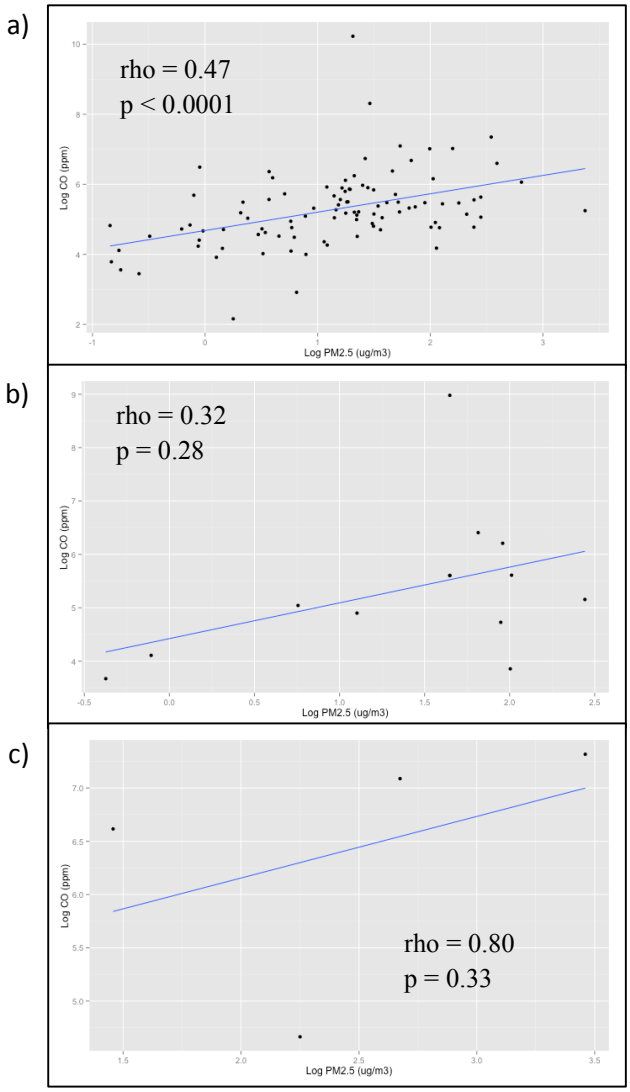
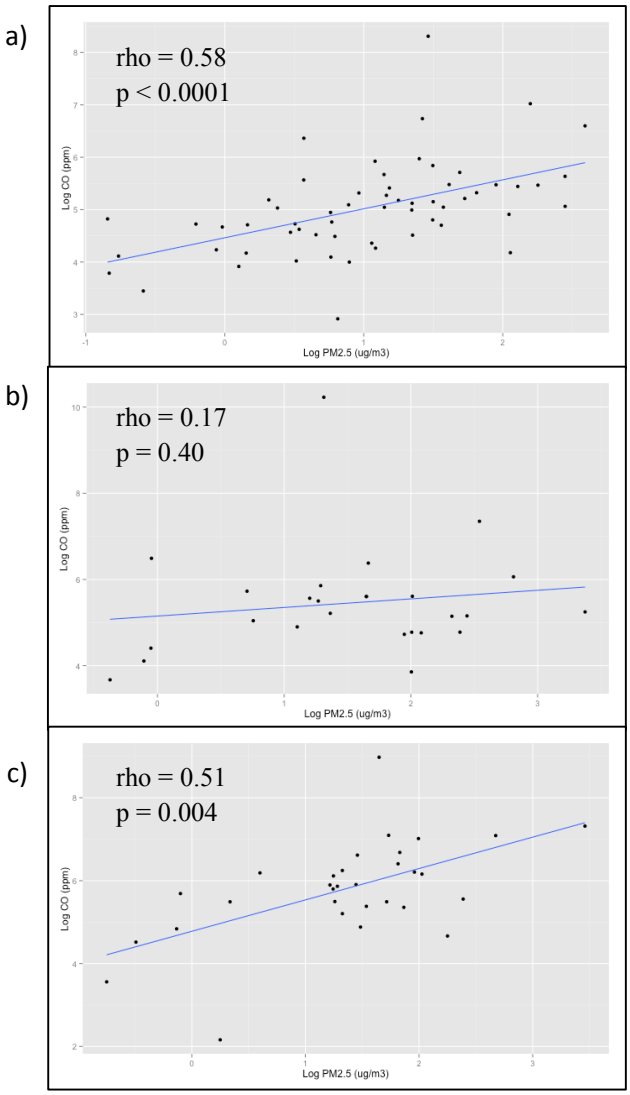


Figure 3.9. Correlation between log-transformed mean 24-hour $PM_{2.5}$ concentration ($\mu g/m^3$) and mean 24-hour CO concentration (ppm) among 117 low-income urban Indian households by composite cooking fuel (a) LPG only, b) Kerosene (No Wood), and c) Any Wood)



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CHAPTER 4

Exposure to secondhand tobacco smoke and validity of exposure assessment questions in women and children in low-income households in India

Abstract

Background: Exposure to secondhand tobacco smoke (SHS) is most often measured through participant report, however valid questions are needed to insure accuracy of measurement. We aimed to assess validity of reported measures of exposure to SHS among adult women and children in low-income urban Indian households.

Methods: A structured questionnaire was developed from published literature, translated into Marathi language, and administered to adult women and the caregivers of included children. Air nicotine monitors were placed in a common living space of each home for a period of 7 days. Hair nicotine analysis was conducted on hair samples from participants who consented to providing a sample. Reported measures of exposure were compared to the environmental and biological measures.

Results: High levels of exposure to SHS were found across the included households. Over 30% of homes (n=32) had detectable air nicotine, and 68% (n=42) of hair samples were found to have detectable levels of nicotine. Correlation between air and hair nicotine concentrations were stronger for children ($r = 0.53$, $p = 0.004$) than adults ($r = -0.98$, $p = 0.78$), however overall low correlation was found. The included questions showed poor correlation with objective measures of exposure, with household smoking rules resulting in the only statistically significant correlation with air nicotine values ($r = 0.55$, $p = 0.007$).

Discussion: Adult women and children in low-income urban Indian communities are highly exposed to SHS, however current methods for assessing exposure using reported measures are inadequate. Additional questions should be developed to more appropriately capture relevant microenvironments and patterns of exposure. When feasible, researchers should use objective environmental and biological markers of nicotine exposure in this population.

Introduction

Exposure to secondhand tobacco smoke (SHS) is an important cause of lung disease in children and non-smoking adults[1]. Assessment for SHS exposure is conducted for population-based prevalence estimates, in epidemiologic studies investigating associations with disease, and by clinicians during patient assessments[2, 3]. Reported measures and questionnaires are the most commonly used methods for assessing SHS exposure levels in populations of interest. These methods are non-invasive, inexpensive, and easy to administer, providing a useful resource to a wide range of professionals for a variety of purposes[3]. Additionally, clinicians and health care professionals often use questionnaire methods for ascertaining exposure among their patients[4].

Of importance, however, is ensuring that questions included in reported measurement tools are valid for their target population. Misclassification of SHS exposure may lead to incorrect prevalence estimates in surveillance studies and biased or confounded estimates of risk in epidemiologic studies, and is frequently cited as a major limitation of studies evaluating SHS exposure[3]. In the clinic setting, patients not identified as being exposed to SHS may miss out on important interventions from their provider. In some studies, environmental and biological samples are collected to confirm reported measures of exposure[5, 6]. However, in large population surveys, biochemical validation of responses is often not feasible for logistical as well as financial reasons. Additionally, the collection of biological samples, such as blood, urine, and saliva, may be deemed as invasive or culturally insensitive in some situations.[7] The validity of reported measurements are an ongoing concern, especially with increased stigma regarding acceptability of tobacco use due to its impact on health.[8-10] As tobacco use becomes more of a socially undesirable behavior, it can be expected that accuracy of self-report for use will change. In general, socially undesirable behaviors have been found to be routinely underreported, and tobacco use and exposure is no exception.[11, 12]

Measurement of exposure to SHS is particularly difficult as individuals may be exposed in a variety of microenvironments, and accurately reporting intensity and duration of exposure is often challenging.[13] A variety of self-report methods have been used to collect SHS exposure information, from daily diaries to directly observed methods.[3] In children, caregivers are often responsible for reporting exposure, which is a challenge if children are not in the presence of their caregiver full-time or if the caregiver themselves is the source and reluctant to accurately report.[14] Children are also a particularly vulnerable population, elevating the importance of accurate classification in this group. The validation of self-reported measures is necessary as accuracy of self-report may vary across populations and cultures. As long as a majority of our tobacco-use and exposure data is collected via self-reported measures, on-going validation of questions will be an important exercise.[7]

To date, there is limited evidence of validation of reported measures for SHS exposure in India[3, 7]. Questions used in the Global Tobacco Surveillance System surveys, which have been administered in India, are validated for understanding and comprehension of questions, but routine validation with biomarkers is not conducted (as is the case with the NHANES survey in the U.S.)[15, 16]. In India, validation of SHS exposure questions in low-income individuals have not been thoroughly conducted, and may provide value to researchers and clinicians evaluating tobacco use in this particularly vulnerable population. The purpose of this present study is to evaluate the validity of reported measures of exposure to tobacco use in India, specifically women's ability to report personal SHS exposure and caregiver ability to report SHS exposure for children less than five years of age.

Methods

The present study was nested within on-going research investigating the association between SHS exposure and tuberculosis (TB) in Pune, India. Ethics approval was granted from the Sassoon General Hospital and Byramjee Jeejeebhoy Medical College (SGH/BJMC) IRB in Pune, India and the Johns Hopkins University IRB. All adult participants, or participant primary caregivers for children, gave written informed consent for participation prior to participation in the parent study.

The details of participant recruitment and sample collection have been previously described (Dissertation Chapter 2). Briefly, eligible participants were recruited from low-income communities served by SGH/BJMC. In the home, field staff administered a structured questionnaire to the participant or their primary caregiver to assess reported measures of personal tobacco use or exposure to SHS. As all children were under 5 years of age, no personal tobacco use questions were administered to them under the assumption that it is unlikely they would be using tobacco products as it was assumed they were too young to be using tobacco products themselves and that exposure to SHS was a more important indicator for this population. The research team placed one passive air nicotine monitor in the common living space of each home, which was left for a period of seven days. For quality control purposes, a 10% sample of blanks and a 10% sample of duplicates were included. Additionally, one small sample of hair was taken from each consenting participant. Approximately 100 strands were cut near the hair root from the back of the scalp, and the three centimeters of hair were analyzed, representing the previous 3 months growth and exposure. Samples were analyzed at the JHSPH Secondhand Smoke Exposure Assessment Laboratory in Baltimore, MD, USA.

Reported Measures

Structured questionnaires were developed to assess reported measures of tobacco exposure. Questionnaires assessing patterns of exposure both in the home and outside the home were administered to each participant, or their primary caregiver for the pediatric participants, at the baseline visit. All questionnaires were translated into Marathi in order to ensure that subjective questions would be asked in a standardized way, with response options that were clear to the respondents, as well as to ensure that questions intended for validation exercises that were taken from existing published sources were true to their intention. Questionnaires were first translated into Marathi by two members of the study team. Next, a third member of the study team with strong English literacy and an Indian Masters student fluent in English back-translated the questionnaires. The study team then compared the back-translated version to the original English version; errors were identified, discussed, and changes based on study team consensus were made in the Marathi version. Minor changes to some of the words and response options were made to accommodate cultural understanding and local situations. An additional independent counselor then read both the English and the Marathi versions to double check for errors. Consensus was made on any disagreements or errors, and a clean "fair copy" of the Marathi translation was developed. An independent third party first typed up this translation, which was reviewed by members of the research team for any errors and adjustments that needed to be made. Certificates of translation were acquired for the translated Marathi version from an outside party.

Questions included in the structured questionnaire were sourced from recommended questions from published literature, including a review of SHS exposure questions by Avila-Tang *et al.* for both children and adults, questions recommended by the American Academy of Pediatrics (AAP) for children, and questions from the Global Adult Tobacco Control Survey (GATS) for the adult women[3, 16, 17]. For some of the questions, a micro-environmental model of exposure was developed to capture information about where the participant spent their time during the previous

7 days. A list of the questions included and their source reference are found in Table 4.1. Small adjustments were made to account for cultural relevance and nuances in language.

Statistical analysis

Descriptive statistics for air nicotine and hair nicotine values were calculated, including median (IQR), range, and whether values were over the limit of detection for the laboratory analysis (0.036 $\mu g/m^3$ for air samples and 0.227 ng/mg for hair samples). Categorical variables were next generated for each marker, with below the limit of detection as the reference category, and two additional categories dichotomized by the median value among those with detectable levels of nicotine. Correlation between air and hair nicotine values to investigate importance of household SHS exposure was estimated using the Spearman Rank test for log-transformed continuous concentrations. Categorical variables were tested for correlation using Spearman Rank test and polychoric correlation, and binary measures of detectable nicotine were estimated using Pearson correlation.

Reported measures were compared with objective air and hair nicotine values using tetrachoric correlation for binary measures of nicotine and polychoric correlation for categorical measures of nicotine. Percent agreement and sensitivity and specificity were also calculated. Reported measure questions that performed well were selected for inclusion in a composite variable, which was also compared with the objective nicotine markers as described above. Statistical analysis was conducted in R (Version 3.2.2)[18].

Results

In total, 97 households consisting of 57 pediatric participants and 40 adult participants were included in this analysis. Air nicotine measurements are included for all households, however 7 (12%) child participants and 16 (40%) adult participants refused hair sample collection. Additionally, 2 (4%) child participants did not have sufficient hair for a sample to be taken. Demographic and socioeconomic characteristics of included participants are presented in Table 4.2. Participants came from low-income communities, with 78% reporting an income less than 15,000 INR (approximately $230 US Dollars) per month and 16% reporting any food insecurity within the last 30 days. None of the adults reported any current tobacco smoking, however 9 (23%) reported current smokeless tobacco use. This exposure would be captured in hair nicotine, but not air nicotine, concentration results.

Air and hair nicotine concentrations for all participants, as well as child and adult participants separately, are listed in Table 4.3. In total, 31 (32%) of homes were found to have detectable levels of air nicotine. This includes 13 (n=23%) of pediatric homes and 18 (45%) of adult homes. A larger proportion of detectable levels were found in hair samples, with 42 (68%) of all participants having detectable levels of hair nicotine. This included 36 (75%) pediatric participants and 5 (60%) of adult participants. As hair samples represent the previous three months of exposure to tobacco both inside and outside of the home, as well as the fact that smokeless tobacco use among women would register on hair nicotine, but not air nicotine values, it is expected that a higher proportion of values would be detectable as compared to household air nicotine.

Correlation between continuous log-transformed air and hair nicotine values are presented in Figure 4.1, and correlation coefficients are presented in Table 4.4. The correlation between log-transformed air and hair nicotine values was statistically significant in children (r =0.39,

p=0.006), however no association was found in the adults, which suffered from a small sample size due to hair sample refusals. This correlation relationship among children did not hold with results dichotomized into detectable and undetectable, however remained significantly correlated with categorical measures of exposure ($r = 0.5$, $p = 0.004$).

Poor correlation was found between reported measures of exposure and both air and hair nicotine results for pediatric participants (Table 4.5). No survey questions were found to be correlated with detectable hair nicotine levels. Questions about where people smoked in the home and household smoking rules were significantly correlated with detectable air nicotine values ($r=0.52$, $p=0.01$ and $r=0.55$, $p=0.007$, respectively). Sensitivity and specificity for self-reported exposure questions were low, especially for hair nicotine comparisons. Questions regarding household smoking patterns and rules were found to have the highest sensitivity (Table 4.6). When questions were combined into a composite question, sensitivity increased to 0.77 (95% CI: 0.46, 0.95) for air nicotine comparisons and 0.53 (95% CI: 0.35, 0.70) for hair nicotine comparisons, but at the cost of specificity for both.

Results of correlations between reported measures and air and hair nicotine values for adults are shown in Table 4.7, however caution should be used during interpretation due to the low number of hair nicotine samples. All self-reported smokeless tobacco users had detectable levels of nicotine in their hair, and percent agreement between reported use and detectable hair nicotine was 79%. Reporting strong household smoking rules was not statistically associated with detectable household air nicotine and the magnitude of correlation was lower than was found in pediatric participant homes ($r=0.29$, $p=0.56$). Whether or not people reported smelling other people prepare mishri at least once per week was also not correlated with air nicotine values ($r=0.09$, $p=0.75$). Similarly, sensitivity and specificity of these reported measures for detectable

air and hair nicotine was low (Table 4.8). The highest sensitivity for air nicotine exposures was for reported weekly exposure to mishri preparation (0.56; 95% CI: 0.31, 0.78) and when questions were combined into a composite measure (0.61; 95% CI: 0.36, 0.83).

Common pediatric and adult questions included household smoking rules and whether or not they smelled the preparation of mishri at least weekly. The correlation of these questions with air and hair nicotine results were low and not statistically significant (Table 4.9 and 4.10).

Discussion

Here we present results of a validation exercise for reported measures of tobacco exposure in a low-income setting in urban India. Air nicotine samples, collected from every home, and hair nicotine samples, collected from those participants consenting to provide hair samples, were compared to each other and to reported measures of exposure to SHS. We found a moderate correlation between log-transformed and categorical air and hair nicotine values in children, as well as moderate correlation between household smoking rules and air nicotine in children. Correlation between reported measures and air and hair nicotine values in adult participant households was low, however caution should be used during interpretation due to low numbers of hair nicotine samples for adults.

Few studies have evaluated the validity of reported measures of tobacco exposure in Indian populations. Self-reported tobacco use among Indian youth in slums in India found low sensitivity (36.3%) for self-reported tobacco use, although SHS exposure was not considered[19]. A second study evaluated reported measures of exposure to SHS with blood cotinine among industrial workers in India. While cotinine, a short-term biomarker for exposure to SHS was used

instead of hair nicotine, the results of the study also indicate that reported measures perform poorly in assessing exposure to SHS among non-smokers[20]. Correlation between hair and air nicotine concentrations are consistently found to be higher in younger children as compared to older children or adults[21]. In a study among women and children in 31 countries living in households with at least one smoker, correlation between household air and hair nicotine was 0.36 ($p<0.001$) for children and 0.25 ($p<0.001$) for adults[22]. Our results are consistent with these findings.

A large proportion of individuals in our study (68%) had detectable levels of nicotine in their hair. It is difficult to translate hair nicotine concentrations into precise units of exposure, such as number of cigarettes per day, due to differences in nicotine metabolism across race and age, as well as type of tobacco product exposure[23]. Several studies, however, have published suggested cut-offs and levels of nicotine found in self-reported tobacco users and SHS exposure at the household level[24]. Kintz et al suggests a cut-off of 2 ng/mg hair nicotine concentration for adult smokers[25]. A review by Avila-Tang et al (2012) identified a cut-off of 0.8ng/mg for non-smoking adults as exposed to SHS[5]. Klein et al reported that adult women reporting exposure to SHS had an average hair nicotine concentration of 3.32 (standard error: 0.85), and those not reporting SHS exposure an average concentration of 1.24 (SE: 0.39)[26]. A study among adults in Baltimore, Maryland, reported median hair nicotine concentrations of 0.23 (IQR: 0.08-0.44) among non-smokers, 0.36 (IQR: 0.27-3.03) among self-reported as exposed to SHS, and 16.2 (4.0-40.6) among active smokers. In this study, a cut-off of 2.77 ng/mg was calculated for distinguishing between smokers and non-smokers [24].

Studies in Indian populations are challenging in that there is a high prevalence of smokeless tobacco use, which will also contribute to biological measures of nicotine exposure. Over 20% of

our adult participants reported smokeless tobacco use, consistent with prevalence estimates reported by the WHO[27]. Removing these individuals from hair nicotine concentration summary statistics lowers the median concentration of nicotine in hair to 0.23 (IQR: 0.23, 0.35). Even so, 30% (n=3) of those reporting no smokeless tobacco use still have detectable levels of hair nicotine, approaching levels seen among women in Asia who live in a household with at least one smoker (median hair nicotine concentration of 0.50 ng/mg)[22].

Hair nicotine concentrations among the children in this study were often higher than other studies reporting hair nicotine concentrations in young children. The median level of hair nicotine found in children in this present study (1.84 ng/mg; IQR: 0.29, 4.23) is higher than was found by Al-Delaimy et al. (2000) among children living in homes with two smokers (median 1.46 ng/mg; IQR: 0.75 – 2.75) but less than those living in homes with more than two smokers (median 2.02 ng/mg, IQR: 1.08 – 4.41)[28]. Kim et al (2009) report median hair nicotine concentrations of 0.80 ng/mg (IQR: 0.27-2.24) among children living in homes with smokers. In a study reporting among children living in households with a smoker in Asia, median hair nicotine concentrations were found to be 0.87 ng/mg, which increased to 1.21 ng/mg (IQR: 0.36, 3.43) when restricting to children under 6 years of age[21]. As all of the child participants in this present study were under 5 years of age, it is unlikely that personal tobacco use, either smoked or smokeless, significantly contributes to hair nicotine concentrations. The case may be, however, that thirdhand tobacco smoke, or airborne nicotine that has settled and ingested orally through hand-to-mouth behavior in young children or through dermal absorption, may contribute to exposure[29]. For young children, Avila-Tang suggests a cut-off of 0.2 ng/mg for children exposed to SHS[5].

Air nicotine concentrations are subject to similar limitations as hair nicotine in terms of variability in results based on type of tobacco exposure[25]. Further, the concentrations found are a

time-weighted average of the duration a monitor is placed in a home, and cannot distinguish between constant low-levels of exposure and one extremely high level. A lower proportion of air nicotine monitors were found to be detectable as compared to hair nicotine values. Air nicotine concentrations in Asian homes of at least one smoker have been reported as 0.09 ug/m^3, which is the median value of those with detectable levels in this present study (0.093 ug/m^3; IQR: 0.01 - 0.21)[22].

Additional research is needed to determine more appropriate questions related to SHS exposure in this vulnerable population. Micro-environmental models of exposure are recommended for reported measures of exposure, and those currently recommended may not be applicable in this population[30]. Childcare may often consist of time spent at family or neighboring households, as compared to day care settings as often seen in higher-income countries. Additionally, restaurants or work settings maybe not be as relevant for individuals living in conditions of extreme poverty, such as the slum areas of urban India. Qualitative research identifying other potentially important locations are needed. Additionally, given the highly polluted settings in which these individuals live, it may be difficult for individuals to recall tobacco-specific pollution (Dissertation Chapter 3).

The results of this study should raise caution to those using or evaluating reported measures of exposure to SHS in these communities in population-based epidemiologic studies and, perhaps more importantly, studies of exposure-disease relationships. Statistical models including self-reported SHS exposure as primary exposure of interest, or as a control variable for a different relationship of interest, may misclassify individual exposure, and readers should be cautious when interpreting the results. When feasible, objective measures of exposure should be used.

Table 4.1. List of exposure questions used and their source references.

Question	Source Reference
Children	
Over the past 7 days, has your child been around secondhand tobacco smoke? Do you remember smelling cigarette, bidi, or hookah/waterpipe smoke when your child was present?	AAP, Avila-Tang 2012
Over the past 7 days, did you and your child visit other people's homes? If yes, did you smell cigarettes, bidis, or hookah?	AAP
Over the past 7 days, did you and your child visit markets, restaurants or other public places? If yes, did you smell cigarettes, bidis, or hookah?	AAP
Over the past 7 days did you and your child visit public transportation (bus or auto rickshaw)? If yes, did you smell cigarettes, bidis, or hookah?	AAP
Over the past 7 days did you and your child visit your place of work? If yes, did you smell cigarettes, bidis, or hookah?	AAP
Does anyone who currently live in your home use mishri?	N/A
If someone who currently lives in your home uses mishri, over the past 7 days, about how many times has mishri been prepared in your home?	N/A
How many people who currently live in your home smoke cigarettes or bidis?	AAP
NOT including yourself, which of the following people living in your home currently smoke cigarettes or bidis (all that apply): a) Your spouse or significant other b) Your child under age 18 c) Other adults in the home	AAP
Does anyone who lives in your home currently use a hookah or waterpipe to smoke tobacco?	AAP
Over the past 3 months, has anyone smoked anywhere inside your home?	AAP
Where do people smoke when they are at your home? a) Inside only b) Inside and outside c) Outside only d) Depends on the season e) No one (including visitors) smokes at my home	AAP
How often does anyone, including visitors, smoke cigarettes or bidis inside your home? a) Daily b) Weekly c) Monthly d) Sometimes, but not every month e) Never	AAP
Please tell me which best describes how cigarette/bidi smoking is handled in your home (home includes proximate outdoor spaces). a) No rules b) Smoking is permitted anywhere c) Smoking is permitted in some places or at some times d) No one is allowed to smoke anywhere	AAP
In general, how often can you smell other people or families preparing mishri when you were inside your own home?	N/A
Adults	

Do you currently smoke tobacco on a daily basis, less than daily, or not at all?	GATS
The next questions are about using smokeless tobacco, such as mishri, gutka, khaini, or betel or pan with tobacco. Smokeless tobacco is tobacco that is not smoked, but is sniffed through the nose held in the mouth, or chewed. Do you currently use smokeless tobacco on a daily basis, less than daily, or not at all?	GATS
Which of the following best describes the rules about smoking inside of your home: Smoking is allowed inside of your home, smoking is generally not allowed inside of your home but there are exceptions, smoking is never allowed inside of your home, or there are no rules about smoking in your home.	GATS
Do you currently work outside of your home? Do you usually work indoors or outdoors? Are there any indoor areas at your work place? Which of the following best describes the indoor smoking policy where you work: Smoking is allowed anywhere, smoking is allowed only in some indoor areas, smoking is not allowed in any indoor areas, or there is no policy?	GATS
Do you currently work outside of your home? Do you usually work indoors or outdoors? Are there any indoor areas at your work place? During the past 30 days, did anyone smoke in indoor areas where you work?	GATS
During the past 30 days, did you visit any restaurants? Did anyone smoke inside of any restaurants that you visited in the past 30 days?	GATS
During the past 30 days, did you use any public transportation? Did anyone smoke inside of any public transportation that you used in the past 30 days?	GATS
In general, how often can you smell other people or families preparing mishri when you were inside your own home?	GATS

AAP = American Academy of Pediatrics
GATS = Global Adult Tobacco Survey

Table 4.2. Demographic and socioeconomic status characteristics among women (n=40) and children (n=57) in low-income households assessed for secondhand smoke exposure in an urban Indian setting.

	Total (n=97)	**Children (n=57)**	**Adults (n=40)**
Family type, n(%)			
Nuclear	47 (48)	32 (56)	15 (38)
Joint	36 (37)	15 (26)	12 (53)
Extended	14 (14)	10 (18)	4 (10)
Head of household is male, n(%)	38 (39)	12 (21)	26 (65)
Years of Education of Head of Household, n(%)			
< 4 years (primary)	29 (30)	18 (32)	11 (28)
≥ 4 years	68 (70)	39 (68)	29 (73)
Employment status of head of household, n(%)			
Skilled/trained worker, housewife, or retired	54 (56)	25 (44)	29 (73)
Unskilled manual worker or unemployed	43 (44)	32 (56)	11 (28)
Monthly household income, n(%)			
≤ 15,000 INR	76 (78)	44 (77)	32 (80)
> 15,000 INR	21 (22)	13 (23)	8 (20)
Religion, n(%)			
Hindu	79 (81)	49 (86)	30 (75)
Other	18 (19)	8 (14)	10 (25)
Reported food insecurity[¥], n(%)	16 (16)	10 (18)	6 (15)
Crowding[π], n(%)	48 (49)	25 (44)	23 (58)
Number of rooms (excluding bathroom), median (IQR)	2 (1, 2)	1 (1, 2)	2 (1, 2)
Number of assets owned[†], median (IQR)	9 (7, 10)	8 (6, 10)	9 (8, 10.3)
Roofing material plastic, rudimentary, or metal sheet[*f*], n(%)	54 (56)	37 (65)	17 (43)
Exterior walls made of bamboo/mud or metal sheets[*f*], n(%)	25 (26)	21 (37)	4 (10)
Flooring concrete or rudimentary (no tiles), n(%)	37 (38)	28 (49)	9 (23)
Electricity supply shared/illegal/none, n(%)	28 (29)	19 (33)	9 (23)
Own toilet facility, n(%)	50 (52)	28 (49)	22 (55)
Health insurance, n(%)	15 (15)	5 (9)	10 (25)
Life Insurance, n(%)	33 (34)	18 (32)	15 (38)
Bank account, n(%)	80 (82)	46 (81)	34 (85)

¥ As measured by the Household Food Insecurity Assessment Survey (HFIAS). Scores ≥ 1 categorized as insecure.
π Greater than the median number of people per room in this sample (median = 3.25 people/room)
f Compared to brick, concrete, or wood
† Assets: Clock/watch, radio, TV, bicycle, mobile phone, mattress or sleeping pad, chair, bed or cot, table, refrigerator, motorcycle, electric fan, car, washing machine, pressure cooker, and sewing machine.

Table 4.3. Air nicotine and hair nicotine concentrations among adult women (n=40) and children (n=57) in low-income communities of Pune, India.

	Pediatric	Adult	All
AIR NICOTINE	**(n=57)**	**(n=40)**	**(n=97)**
Air Nicotine ($\mu g/m^3$), median (IQR)	0.001 (0.001, 0.001)	0.001 (0.001, 0.01)	0.001 (0.001, 0.01)
Air Nicotine ($\mu g/m^3$), min, max	0.001, 1.97	0.001, 2.02	0.001, 2.02
Air Nicotine Detectable, n(%)	13 (23)	18 (45)	31 (32)
Air Nicotine Geometric Mean (SD)	0.003 (10.4)	0.004 (7.5)	0.004 (9.1)
Air Nicotine, categorical¥			
Undetectable	44 (77)	22 (55)	75 (77)
Low detectable	7 (12)	9 (23)	7 (7)
High detectable	6 (11)	9 (23)	15 (15)
HAIR NICOTINE	**(n=48)**	**(n=14)**	**(n=62)**
Hair Nicotine (ng/mg), median (IQR)	1.84 (0.29, 4.23)	0.23 (0.23, 8.65)	1.35 (0.23, 4.30)
Hair Nicotine (ng/mg), min, max	0.03, 19.79	0.23, 13.0	0.03, 19.79
Hair Nicotine Geometric Mean (SD)	1.06 (7.06)	0.90 (6.3)	1.02 (6.79)
Hair Nicotine Detectable, n(%)	36 (75)	5 (60)	42 (68)
Hair Nicotine, categorical†			
Undetectable	12 (25)	8 (57)	20 (32)
Low detectable	18 (38)	3 (21)	21 (34)
High detectable	18 (38)	3 (21)	21 (34)

Note: 7 child participants refused to give a hair sample, and 2 child participants did not have sufficient hair for sample. 16 adult participants refused to give hair, and analysis for the remaining samples are pending due to laboratory delays.

¥ Air nicotine categories for children: Undetectable (≤ 0.0029), Low detectable (0.003 – 0.19), High detectable (> 0.19); Air nicotine categories for adults: Undetectable (≤ 0.0029), Low detectable (0.003 – 0.021), High detectable (> 0.021)

† Hair nicotine categories for children: Undetectable (≤ 0.227), Low detectable (0.228 – 2.69), High detectable (> 2.69); Air nicotine categories for adults: Undetectable (≤ 0.227), Low detectable (0.228 – 10.89), High detectable (> 10.89)

Figure 4.1. Scatter plots of air nicotine and hair nicotine concentrations in homes of a) children (n=57) , b) adult women (n=40), c) children and adult women combined (n=97) in low-income communities of Pune, India.

a)

b)

c)

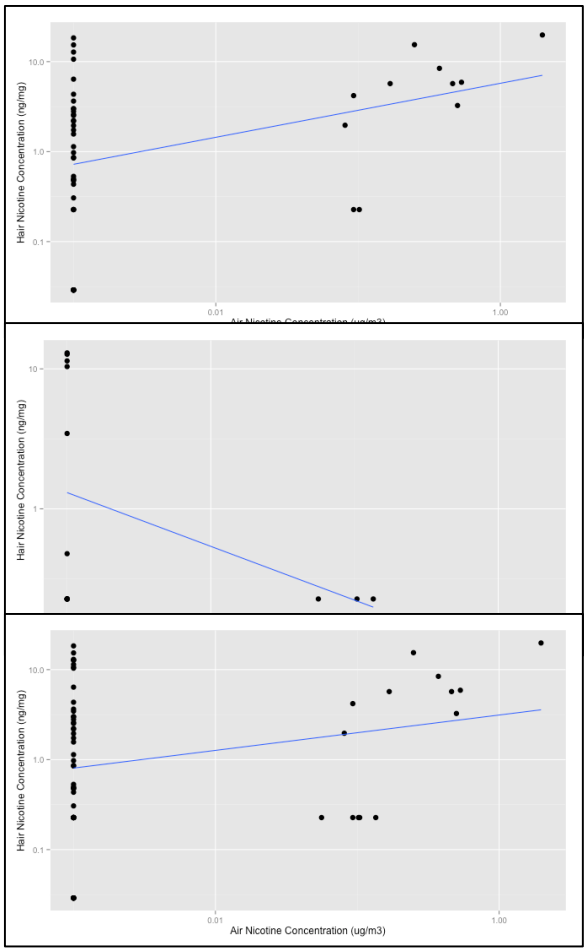

Table 4.4. Correlation of air nicotine and hair nicotine concentrations in homes of children (n=57), adult women (n=40), and children and adult women combined (n=97) in low-income communities of Pune, India.

	Continuous		**Dichotomous**		**Categorical**	
	Correlation (r)[¥]	**p-value**	**Correlation (r)[†]**	**p-value**	**Correlation (r)[Δ]**	**p-value**
Children	**0.39**	**0.006**	0.17	0.66	**0.53**	**0.004**
Adult	-0.43	0.13	-0.98	0.78	-0.98	0.78
Both	0.21	0.11	-0.08	0.76	0.27	0.38

*Bolded values statistically significant at $p < 0.05$
¥ Spearmak Rank
† Tetrachoric Correlation
Δ Polychoric Correlation

Table 4.5. Percent agreement and correlation for reported measures of exposure to secondhand smoke and air and hair nicotine concentrations in children (n=57) in low-income communities of Pune, India.

		Air Nicotine			Hair Nicotine		
	n(%)	% Agreement	Corr.	p-value*	% Agreement	Corr.	p-value*
Over the past 7 days, has your child been around SHS?	22 (39)	63	0.29	0.33	46	0.09	0.75
Over the past 7 days did your child visit other people's homes, and did you smell cigarettes, bidis, or hookah?	7 (12)	--	--	--	25	-0.41	0.24
Over the past 7 days did your child visit markets, restaurants or other public places, and did you smell SHS?	11 (20)	--	--	--	32	-0.03	0.79
Over the past 7 days did your child visit public transportation (bus or auto rickshaw), and did you smell SHS?	10 (18)	--	--	--	38.3	0.96	0.80
Over the past 7 days did your child visit your place of work, and did you smell SHS?	2 (4)	--	--	--	--	--	--
Does anyone who currently live in your home use mishri?	19 (34)	64	0.24	0.45	43.8	14	0.68
If someone uses mishri, over the past 7 days about how many times has mishri been prepared in your home?– positive response is anything greater than 0	6 (33)	63	0.25	0.66	33	0.90	0.79
How many people who currently live in your home smoke cigarettes or bidis? (At least one person smokes)	21 (37)	58	0.03	0.79	46	0.09	0.75
Not including yourself, which of the following people living in your home currently smoke cigarettes or bidis?							
Your spouse or significant other	11 (19)	72	0.29	0.39	33	-0.11	0.73
Your child under age 18	0 (--)	--	--	--		--	--
Other adults in the home	10 (18)	67	-0.06	0.76	37.5	0.29	0.49
Does anyone who lives in your home currently use a hookah or waterpipe to smoke tobacco?	4 (7)	70	-0.89	0.79	33	0.90	0.79

Over the past 3 months, has anyone smoked anywhere inside your home?	18 (32)	67	0.29	0.34	35	-0.29	0.36
Where do people smoke when they are at your home? (outside/none vs inside)	15 (26)	72	0.41	0.12	33	-0.20	0.59
Where do people smoke when they are at your home? (none vs inside/outside)	23 (40)	68	**0.52**	**0.01**	46	0	0.80
How often does anyone, including visitors, smoke cigarettes or bidis inside your home? (≤ monthly vs daily/weekly)	7 (12)	75	0.35	0.32	31	-0.07	0.78
Household smoking rules (not allowed vs sometimes-no rules)	22 (39)	70	**0.55**	**0.007**	48	0.04	0.79
Combining exposure questions (exposed to SHS, spouse smoke, household smoking rules)	29 (51)	61	**0.49**	**0.03**	52	0.04	0.78

*Bolded values are statistically significant at $p < 0.05$

Table 4.6. Sensitivity and specificity of reported measures of exposure to secondhand smoke as compared to air and hair nicotine concentrations in children (n=57) in low-income communities of Pune, India.

		Gold Standard							
		Air Nicotine				**Hair Nicotine**			
	n(%)	**Sens (%) (95% CI)**	**Spec (%) (95% CI)**	**PPV (%) (95% CI)**	**NPV (%) (95% CI)**	**Sens (%) (95% CI)**	**Spec (%) (95% CI)**	**PPV (%) (95% CI)**	**NPV (%) (95% CI)**
Over the past 7 days, has your child been around SHS?	22 (39)	0.54 (0.25, 0.81)	0.66 (0.50, 0.80)	0.32 (0.14, 0.55)	0.83 (0.66, 0.93)	0.39 (0.23, 0.57)	0.67 (0.35, 0.90)	0.78 (0.52, 0.94)	0.27 (0.12, 0.46)
Over the past 7 days did your child visit other people's homes, and did you smell cigarettes, bidis, or hookah?	7 (12)	--	--	--	--	0.08 (0.02, 0.22)	0.75 (0.43, 0.95)	0.50 (0.12, 0.88)	0.21 (0.10, 0.37)
Over the past 7 days did your child visit markets, restaurants or other public places?	11 (20)	--	--	--	--	0.17 (0.06, 0.33)	0.82 (0.48, 0.98)	0.75 (0.35, 0.97)	0.23 (0.11, 0.39)
Over the past 7 days did your child visit public transportation (bus or auto rickshaw)?	10 (18)	--	--	--	--	0.19 (0.08, 0.36)	1.00 (0.62, 1.00)	1.00 (0.47, 1.00)	0.28 (0.15, 0.44)
Over the past 7 days did your child visit your place of work?	2 (4)	--	--	--	--	--	--	--	--
Does anyone who currently live in your home use mishri?	19 (34)	0.46 (0.19. 0.75)	0.70 (0.54, 0.83)	0.32 (0.13, 0.57)	0.81 (0.65, 0.92)	0.33 (0.19, 0.51)	0.75 (0.43, 0.95)	0.80 (0.52, 0.96)	0.27 (0.13, 0.46)
If someone uses mishri, over the past 7 days about how many times has mishri been prepared in your home? Median (IQR) – positive response is anything greater than 0	6 (33)	0.33 (0.04, 0.78)	0.80 (0.44, 0.97)	0.50 (-.07, 0.93)	0.67 (0.35, 0.90)	0.20 (0.03, 0.56)	1.00 (0.09, 1.00)	1.00 (0.09, 1.00)	0.20 (0.03, 0.56)
How many people who currently live in your home smoke cigarettes or bidis? (At least one person smokes)	21 (37)	0.38 (0.14, 0.68)	0.64 (0.48, 0.78)	0.24 (0.08, 0.47)	0.78 (0.61, 0.90)	0.39 (0.23, 0.57)	0.67 (0.35, 0.90)	0.78 (0.52, 0.94)	0.27 (0.12, 0.46)

Not including yourself, which of the following people living in your home currently smoke cigarettes or bidis?									
Your spouse or significant other	11 (19)	0.31 (0.09, 0.61)	0.84 (0.70, 0.93)	0.36 (0.11, 0.69)	0.80 (0.66, 0.91)	0.19 (0.08, 0.36)	0.75 (0.43, 0.95)	0.70 (0.35, 0.93)	0.24 (0.11, 0.40)
Your child under age 18	0 (--)	--	--	--	--	--	--	--	--
Other adults in the home	10 (18)	0.15 (0.02, 0.45)	0.82 (0.67, 0.92)	0.20 (0.03, 0.56)	0.77 (0.62, 0.88)	0.19 (0.08, 0.36)	0.92 (0.62, 1.00)	0.88 (0.47, 1.00)	0.28 (0.15, 0.44)
Does anyone who lives in your home currently use a hookah or waterpipe to smoke tobacco?	4 (7)	0 (0.00, 0.34)	0.91 (0.78, 0.97)	0.00 (0.00, 0.72)	0.75 (0.62, 0.86)	0.11 (0.03, 0.26)	1.00 (0.64, 1.00)	1.00 (0.28, 1.00)	0.27 (0.15, 0.43)
Over the past 3 months, has anyone smoked anywhere inside your home?	18 (32)	0.46 (0.19, 0.75)	0.73 (0.57, 0.85)	0.33 (0.13, 0.59)	0.82 (0.66, 0.92)	0.31 (0.16, 0.48)	0.50 (0.21, 0.79)	0.65 (0.38, 0.86)	0.19 (0.07, 0.37)
Where do people smoke when they are at your home? (outside/none vs inside)	15 (26)	0.46 (0.19, 0.75)	0.80 (0.65, 0.90)	0.40 (0.16, 0.68)	0.83 (0.69, 0.93)	0.22 (0.10, 0.39)	0.67 (0.35, 0.90)	0.67 (0.35, 0.90)	0.22 (0.10, 0.39)
Where do people smoke when they are at your home? (none vs inside/outside)	23 (40)	0.69 (0.39, 0.91)	0.68 (0.52, 0.81)	0.39 (0.20, 0.61)	0.88 (0.73, 0.97)	0.42 (0.26, 0.59)	0.58 (0.28, 0.85)	0.75 (0.51, 0.91)	0.25 (0.11, 0.45)
How often does anyone, including visitors, smoke cigarettes or bidis inside your home? (≤ monthly vs daily/weekly)	7 (12)	0.23 (0.05, 0.54)	0.91 (0.78, 0.97)	0.43 (0.10, 0.82)	0.80 (0.66, 0.90)	0.14 (0.05, 0.29)	0.83 (0.52, 0.98)	0.71 (0.29, 0.96)	0.24 (0.12, 0.40)
Household smoking rules (not allowed vs sometimes-no rules)	22 (39)	0.69 (0.39, 0.91)	0.70 (0.55, 0.83)	0.41 (0.21, 0.64)	0.89 (0.73, 0.97)	0.44 (0.28, 0.62)	0.58 (0.28, 0.85)	0.76 (0.53, 0.92)	0.26 (0.11, 0.46)
Combining exposure questions (exposed to SHS, spouse smoke, household smoking rules)	22 (39)	0.77 (0.46, 0.95)	0.57 (0.41, 0.72)	0.34 (0.18, 0.54)	0.89 (0.72, 0.98)	0.53 (0.35, 0.70)	0.50 (0.21, 0.79)	0.76 (0.55, 0.91)	0.26 (0.10, 0.48)

Table 4.7. Percent agreement and correlation for reported measures of exposure to secondhand smoke and air and hair nicotine concentrations in adult women (n=40) in low-income communities of Pune, India.

		Air Nicotine			Hair Nicotine		
	n(%)	**% Agreement**	**Corr.**	**p-value***	**% Agreement**	**Corr.**	**p-value***
Current smoking (daily or less than daily)	0 (--)	--	--	--	--	--	--
Current smokeless tobacco (daily or less than daily)	9 (23)	--	--	--	79	--	--
Household smoking rules (not allowed vs sometimes-no rules)	3 (8)	58	0.29	0.56	64	0.96	0.79
Smoking policy at work (allowed vs not allowed) (among the n = 10 who report working outside of the house)	3 (43)	--	--	--	--	--	--
Past 30 day SHS exposure at work (among n=17)	3 (38)	--	--	--	--	--	--
Past 30 day SHS exposure at restaurants (among n=9)	2 (29)	--	--	--	--	--	--
Past 30 day SHS exposure on public transportation (among n=23)	6 (30)	--	--	--	56	0.96	0.79
How often smell other people preparing mishri (≤ monthly vs daily/weekly)	21 (53)	53	0.09	0.75	43	-0.26	0.65
Combining exposure questions (Household smoking rules and smell mishri)	23 (58)	53	0.10	0.73	50	0.0	0.80

*Bolded values are statistically significant at $p < 0.05$

Table 4.8. Sensitivity and specificity of reported measures of exposure to secondhand smoke as compared to air and hair nicotine concentrations in adult women (n=40) in low-income communities of Pune, India.

		Gold Standard							
		Air Nicotine				**Hair Nicotine**			
	n(%)	**Sens (%) (95% CI)**	**Spec (%) (95% CI)**	**PPV (%) (95% CI)**	**NPV (%) (95% CI)**	**Sens (%) (95% CI)**	**Spec (%) (95% CI)**	**PPV (%) (95% CI)**	**NPV (%) (95% CI)**
Current smoking (daily or less than daily)	0 (--)	--	--	--	--	--	--	--	--
Current smokeless tobacco (daily or less than daily)	9 (23)	--	--	--	--	--	--	--	--
Household smoking rules (not allowed vs sometimes-no rules)	3 (8)	0.11 (0.01, 0.35)	0.95 (0.77, 1.00)	0.67 (0.09, 0.99)	0.57 (0.39, 0.73)	0.17 (0.00, 0.64)	1.00 (0.52, 1.00)	1.00 (0.01, 1.00)	0.62 (0.32, 0.86)
Smoking policy at work (allowed vs not allowed)	3 (43)	--	--	--	--	--	--	--	--
Past 30 day SHS exposure at work	3 (38)	--	--	--	--	--	--	--	--
Past 30 day SHS exposure at restaurants	2 (29)	--	--	--	--	--	--	--	--
Past 30 day SHS exposure on public transportation	6 (30)	--	--	--	--	0.20 (0.01, 0.72)	1.00 (0.28, 1.00)	1.00 (0.01, 1.00)	0.50 (0.16, 0.84)
How often smell other people preparing mishri (≤ monthly vs daily/weekly)	21 (53)	0.56 (0.31, 0.78)	0.50 (0.28, 0.72)	0.48 (0.26, 0.70)	0.58 (0.33, 0.80)	0.33 (0.04, 0.78)	0.50 (0.16, 0.84)	0.33 (0.04, 0.78)	0.50 (0.16, 0.84)
Combining exposure questions (Household smoking rules and smell mishri)	23 (58)	0.61 (0.36, 0.83)	0.45 (0.24, 0.68)	0.48 (0.27, 0.69)	0.59 (0.33, 1.90)	0.50 (0.12, 0.88)	0.50 (0.16, 0.84	0.43 (0.10, 0.82)	0.57 (0.18, 0.90)

Table 4.9. Percent agreement and correlation for reported measures of exposure to secondhand smoke and air and hair nicotine concentrations in adult women and children (n=97) in low-income communities of Pune, India.

		Air Nicotine			Hair Nicotine		
	n(%)	**% Agreement**	**Corr.**	**p-value***	**% Agreement**	**Corr.**	**p-value***
Household smoking rules (not allowed vs sometimes-no rules) (n=101)	25 (26)	65	0.26	0.25	52	0.26	0.36
How often smell other people preparing mishri (≤ monthly vs daily/weekly) (n=51)	23 (49)	55	0.16	0.61	42	-0.23	0.64
Combining exposure questions (no smoking allowed in house and don't smell other's preparing mishri) (n=51)	26 (55)	57	0.26	0.39	53	-0.24	0.64

*Bolded values are statistically significant at $p < 0.05$

Table 4.10. Sensitivity and specificity of reported measures of exposure to secondhand smoke as compared to air and hair nicotine concentrations in adult women and children (n=97) in low-income communities of Pune, India.

		Gold Standard							
		Air Nicotine				**Hair Nicotine**			
	Total, n(%)	**Sens (%) (95% CI)**	**Spec (%) (95% CI)**	**PPV (%) (95% CI)**	**NPV (%) (95% CI)**	**Sens (%) (95% CI)**	**Spec (%) (95% CI)**	**PPV (%) (95% CI)**	**NPV (%) (95% CI)**
Household smoking rules (not allowed vs sometimes-no rules) (n=100)	25 (26)	0.35 (0.19, 0.55)	0.79 (0.67, 0.88)	0.44 (0.24, 0.65)	0.72 (0.60, 0.82)	0.40 (0.26, 0.57)	0.75 (0.51, 0.91)	0.77 (0.55, 0.92)	0.38 (0.23, 0.54)
How often smell other people preparing mishri (≤ monthly vs daily/weekly) (n=50)	23 (49)	0.55 (0.32, 0.77)	0.56 (0.35, 0.75)	0.48 (0.27, 0.69)	0.62 (0.41, 0.81)	0.30 (0.07, 0.65)	0.56 (0.21, 0.86)	0.43 (0.10, 0.82)	0.42 (0.15, 0.72)
Combining exposure questions (no smoking allowed in house and don't smell other's preparing mishri) (n=50)	26 (55)	0.65 (0.41, 0.85)	0.52 (0.32, 0.71)	0.50 (0.30, 0.70)	0.67 (0.43, 0.85)	0.50 (0.19, 0.81)	0.56 (0.21, 0.86)	0.56 (0.21, 0.86)	0.50 (0.19, 0.81)

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CHAPTER 5

Discussion

Summary of Study Findings

The objective of this study was to assess the association of exposure to household air pollution (HAP) with TB in women and children, and further, to characterize these exposures in low-income communities in urban areas of India. Overall, we found extremely high levels of exposure to air pollutants in the home and evidence that HAP may be positively associated with TB in women and children. Specifically:

- **AIM 1**: Low-income households reported a variety of fuel used for cooking in the home, including LPG, kerosene, and wood. While a majority of households use LPG as their primary fuel source, over 40% of homes reported a secondary fuel source, with wood and kerosene highly prevalent. In a multivariate adjusted model, those with TB tended to have higher measures of HAP exposure as compared to healthy controls. This included both reported measures of exposure to household fuel sources, as well as measured concentrations of $PM_{2.5}$ at the primary cooking stove. Importantly, across all study homes, all participants were exposed to extremely high levels of $PM_{2.5}$, regardless of their reported fuel use.
- **AIM 2**: Households in low-income communities in urban India are exposed to high levels of air pollutants, with measures well above the 24-hour recommended limits established by the World Health Organization (WHO). Households primarily used LPG, kerosene, and wood fuel for cooking activities in the home, with nearly 50% of homes reporting secondary fuel sources, most often unclean kerosene and wood fuels. In adjusted analysis, households in the highest quartile of exposure for 24-hour average $PM_{2.5}$ were more likely to report using mosquito coils and to have kitchen areas made of all concrete or brick materials. Households in the lowest quartile of exposure had levels of $PM_{2.5}$ below the WHO interim target of 75 $\mu g/m^3$ for the majority of the day. Identifying characteristics of those in the lowest category, as well as in the highest

exposure category, will help inform intervention strategies for decreasing exposure in this highly vulnerable population.

- **AIM 3**: High levels of exposure to secondhand tobacco smoke were reported among women and children, and were also found in environmental and biological measures of exposure. Hair nicotine concentrations in many of the included participants were at levels found in other studies of individuals living in households with at least one smoker. Correlation between hair and air nicotine concentrations were low and were similar to other studies. No reported measure was found to be a valid measure of exposure, and further research is needed to identify more appropriate questions for administration in these communities.

Implications for Policy and Practice

The results of this study lend weight to the importance of HAP as an important risk factor for TB disease, with implications not only for the Indian Revised National Tuberculosis Control Program (RNTCP), but TB control programs across the globe. Many high-burden TB countries suffer a duel-burden of both TB and HAP exposure, and exposure to HAP may be an important driver of the TB epidemic in countries where large proportions of the population rely on low-efficiency fuels such as biomass and kerosene. While in many countries current TB strategies have begun to halt incidence and have treated millions, new strategies are needed to prevent the still heavy burden of disease facing primarily LMICs. Outside of treatment-as-prevention efforts of TB control programs, current prevention strategies for TB primarily focus on the use of isoniazid preventive therapy for contacts of infectious cases that are at highest risk of transitioning to active disease, mainly children and individuals with HIV infection[1]. Additional prevention efforts are needed, however, as evidenced by the immense worldwide burden of disease. Preventive efforts

also become more important as the emergence of MDR-TB bears more weight on TB control programs[2].

Understanding and mitigating the effect of social determinants of health is a likely frontier in TB Control Program efforts. With the mounting evidence that socioeconomic and environmental factors increase risk for TB, focusing on these alleviating these factors may help ease the TB burden in the most vulnerable to communities[3, 4]. Further, these types of efforts are likely necessary to reach long-term global targets of TB control, especially for elimination by 2050. The Stop TB Strategy currently calls for efforts to mitigate the TB burden among those most vulnerable, specifically identifying individuals in poor communities. Participatory efforts by communities are also highlighted as being an important step in reaching global targets, which may be particularly important in the context of HAP[5]. The results of this study show a great deal of wood use on the exterior of homes, with households reporting only using LPG also reporting exposure from neighboring homes. Interventions to reduce HAP exposure in urban communities have a natural home in the context of community involvement and support to eliminate TB.

Understanding individual and household risk factors for disease may also prove valuable in the detection of additional TB cases. Novel case-finding strategies will likely be a key component of future TB control programs, and identifying those at highest risk will help concentration efforts and most efficiently utilize the limited resources available for supplementary program activities. Current efforts for active case-finding are focusing on the household level, as it is postulated that those living in a home with an active TB case are at highest risk for becoming infected and subsequently transitioning to active disease. In addition to increased likelihood of becoming infected, it is likely that those sharing a living space with an individual with TB have many of the same risk factors as the index case. Exposure to high levels of HAP may be a useful household

characteristic to identify households or communities at greatest risk for having high rates of TB. The use of HAP exposure as a screening tool may prove to be an efficient method for identifying individuals with TB infection or disease.

Further exploration of the potential impact of interventions to reduce HAP and subsequent TB disease is needed. The current gap in funding for the implementation of existing interventions is $1.4 billion, with the highest gaps reported from LMIC[2]. If low-cost interventions can be found to reduce exposure to HAP, these types of interventions may prove cost-effective in reducing burden of disease and subsequently ease the current financial burden of case detection and treatment on TB programs. As HAP is a known risk factor for many diseases, partnerships with other disease control programs may be an efficient way to implement prevention strategies covering multiple diseases. The results of this study lend weight to the importance of HAP exposure in the context of TB, and may be useful in motivating HAP exposure interventions.

These results will also inform modeling exercises, which can provide insight as to the importance of HAP across populations and how decreases in exposure may alter the epidemic over coming decades. Importantly, current estimates of burden of disease do not consider the contribution of morbidity and mortality from HAP-precipitated TB[6]. Additionally, these results will also help inform estimates of the TB burden attributable to HAP. The population attributable fraction for the contribution of HAP to TB had been previously reported at 22%, however the effect size used to calculate this estimate was based on a relative risk of 1.4%, well below the magnitude of effect reported here[4]. Updates to these estimates, with considerations of how exposure is defined, would provide a more accurate understanding, and likely higher population attributable fraction, of the burden of disease from HAP.

Lessons from the effect of tobacco smoking on TB should be heeded in the interpretation of the findings that HAP likely increase risk for TB among those highly exposed. The scientific evidence indicates that those who smoke tobacco, in addition to being at greater risk for TB, may also be at greater risk for negative outcomes following diagnosis[7]. Tobacco smokers may also be less likely to be diagnosed in a timeline manner due to the usualness of coughing, which may not be identified as abnormal until long into the period of infectiousness. Given the similarities in the combustion product constituents, mechanism of exposure, and biological plausibility for disease between tobacco smoking and HAP (SHS included), it should be similar considerations should be given to HAP, as well.

The results of this study should also raise additional concern about the impact of ambient air pollution on TB. In the present study, we use $PM_{2.5}$ as an objective measure of exposure to HAP, and we have raised concerns that neighborhood combustion may be contributing to poor indoor air quality. This marker of exposure may also be used to assess pollution generated from traffic, coal-burning for energy generation, and other sources of ambient air pollution. Given the positive association between $PM_{2.5}$ and TB, policy regulating outdoor air pollution sources is also supported.

Study Limitations

To measure exposure to HAP, exposure was evaluated primarily at the household level, both with reported measures of exposure and the objective measures taken at the primary cook stove. While it is likely that those with higher levels of exposure in their homes also have higher personal measures of exposure, personal monitoring was not conducting and therefore it is unknown if significant exposures were present to the participants outside of their homes. Additionally, to assess the association between HAP and TB, our primary exposure of interest was defined by

measures of $PM_{2.5}$, which was not differentiated by source or composition of the particulate matter. There is evidence that differentiating components of particulate matter may provide a clearer understanding of important components of exposure. For example, combusted kerosene or household refuse may produce harmful chemicals that can adsorb to particulate particles, making them potentially more harmful than the same mass of particulates from combusted wood alone[8]. Variability in this type of exposure may be unaccounted for, leading to incorrect effect sizes should certain components of particulates prove more harmful than others. Misclassification error may also arise from defining exposure using an environmental measurement as opposed to a biomarker, which would better assess internal dose.

The use of $PM_{2.5}$ may also not sufficiently capture exposure to kerosene, as particulate combustion products from this fuel source may be primarily in the ultrafine fraction, and not substantially contribute to the mass concentration as measured by $PM_{2.5}$ measures[9]. Further, this ultrafine fraction may contain components harmful to health, such as chemicals adhered to particulates. Other gaseous emissions from kerosene combustion may also play an important role in increased susceptibility to TB disease, however these were not captured in the current protocol. The importance of kerosene as an exposure is suggested by the effect size seen in the reported measures of exposure, and additional research on kerosene as a primary exposure of interest is needed. Ventilation factors also play an important role in exposure patterns, and subjective reported and observable characteristics were used to control for this aspect.

Environmental and biological markers of exposure to SHS are some of the strongest ways to classify exposure in individuals, and air nicotine and hair nicotine markers are recommended for use for a variety of reasons. As such, they were selected as our standards of exposure for exposure estimation in our participants, as well as to estimate the usefulness of reported measures

of exposure as a metric for true exposure. Despite the strengths of these objective measures, environmental and biological markers of SHS exposure have their own limitations in interpretation. These markers of exposure are indications of exposure over an integrated period of time, and this often did not correspond perfectly with the questions we asked. These measures are also subject to variability due to the nature of field collection and laboratory analysis. Cut-points for exposure classification are not clear, and internal dose as measured by biological markers is not necessarily an indication of toxicity, which may vary between individuals. Despite these limitations, the measures utilized in this study provide robust evidence of a high prevalence of exposure and low levels of correlation between what individuals are able to report and what exposure is measured.

Finally, identifying high-risk groups based on environmental exposure in this population is extremely difficult due to the ubiquitously high exposures we found. Estimating an effect size for an exposure of interest given the extremely high levels of exposure in our "unexposed" group has implications for interpretation. Nevertheless, the results of this study highlight a population extremely vulnerable to both the consequences of HAP exposure, as well as one that is often overlooked due to what is perceived as moderate exposure when compared to those in rural populations. The levels of air pollution that these individuals are exposed to, however, are magnitudes greater than what is recommended as an upper limit. Additionally, urban environments are subject to unique exposures unique in addition to HAP, and often comprising environmental exposures not seen in rural areas. Coupled with environmental exposures, the population density in urban environments may lead to an overall increased impact on disease, especially considering the increased incidence of TB often found in low-income urban communities.

Study Strengths

The current evidence of the association between HAP and TB uses reported measures of exposure to classify individuals and estimate effect sizes. This is the first study to use objective measures of exposure to understand and classify individuals based on household concentrations. Reported measures of exposure were evaluated to provide context and comparison with existing studies, however the use of $PM_{2.5}$ and CO concentrations in the home strengthen the ability to interpret the resulting estimates of association. The use of $PM_{2.5}$ allows interpretation of the results in the context of one of the most commonly measured and regulated pollutants. In addition, CO is commonly measured and will allow for comparisons of this pollutant in the context of other research (both exposure-disease relationships as well as intervention strategies to decrease exposure). The direct-reading instruments used in this study also provided the unique opportunity to investigate how peaks in exposure may influence the exposure-disease relationship. This proved important in our analysis of number of hours greater than 75 $\mu g/m_3$. Without this unique perspective we would be limited in our understanding of the types of exposures found in these communities and what types of strategies may be important for their mitigation.

The use of kerosene as a cooking fuel was also highly prevalent in the community in which this study was conducted. Few existing studies have reported on the use of kerosene as a cooking fuel and its association with TB. The present study supports existing evidence indicating that the combustion of kerosene may increase risk for TB. Further, as kerosene does not appreciably contribute to $PM_{2.5}$ mass measurements, a different pathway or set of exposures unique to the combustion products of kerosene may be implicated. The use of both reported and objectively measured concentrations of exposure in this study allowed for this observation.

Not only were objective measures of air pollution used to classify participant exposure, but robust methods were used to control for SES, which is strongly associated with both TB and the types of fuels that households use. SES is a complex indicator, and is often defined using household income, education or employment status, or asset indexes. This study employed principal component analysis to establish an SES indicator, allowing a variety of variables to potentially play a role in SES, as well as to be included in the analysis. Given the relatively small sample size of this study, it also provided a more robust indicator of SES with fewer variables, resulting in more power to assess the effect size of interest. While this SES indicator cannot be used across varying populations, the primary purpose of this study is to consider HAP and to control for SES, not understand its contribution to risk.

Participants from this study were drawn from an extremely vulnerable population, one with a litany of risk factors for not only TB, but many other health issues. The burden of TB is high in this population, and the stigma around TB is also great. The ability to successfully recruit and conduct household research in this population in the number of households included in this study is one of its great strengths, especially the successful recruitment of community controls. The ability to recruit controls from the same neighborhoods as the included cases provided the ability to control for ambient air pollution, which would have been extremely difficult considering the existing resources necessary to measure ambient air pollution. Although it is likely that ambient air exposure is also a risk factor for TB, the ability to focus on household-level exposure provides insight into more immediate and attainable intervention strategies.

Finally, the inclusion of children in this research is extremely important due to the overall lack of understanding of risk factors in this population, as well as the extreme vulnerability of children to environmental exposures. Children are an often-overlooked population, both in terms of

understanding risk factors of importance, prevention efforts, and treatment options. While IPT is recommended for children in India exposed to an adult with pulmonary TB, this is rarely accomplished. In the context of air pollution, children may be especially vulnerable due to their developing lungs and immune system, as well as elevated respiratory rate. Children may also be more highly exposed to SHS due to the potential exposure pathway from settled tobacco smoke. Recent evidence indicates that thirdhand tobacco smoke, or the exposure to residual settled components of SHS, may be an important exposure pathway, and crawling and hand to mouth behavior may increase these exposures in children. Similar considerations should be made for dermal and/or oral exposure of settled components of combustion of other products as well, especially those of kerosene considering their chemical constituents.

Future Research and Next Steps

While additional research is required to detect a statistically significant association between HAP as measured by $PM_{2.5}$ and TB, the results of this study suggest that HAP exposure is an important risk factor for TB in both adults and children. Future studies should continue to investigate this association using direct-reading measures to ensure the ability to explore both time-weighted averages of exposure as well as the contributions of peaks in exposure. Characterizing exposure levels in these, and other, communities will further identify priority populations, directing intervention efforts to those most vulnerable and at risk.

Additionally, while $PM_{2.5}$ and CO were found at elevated levels, there is a lack of information on other constituents of air pollution, their prevalence in these communities, and their association with TB. For example, $PM_{2.5}$ derived from a variety of sources may contain varying levels of chemical constituents, some which may be more important in the exposure-disease pathway than others. Further characterizing particulate collected in the home will help distinguish the relative

importance of sources. Investigation of the ultrafine fraction and its association with TB will also give a better understanding of the impact of kerosene combustion products. Differentiating particulate composition, and conducting source apportionment exercises, will help determine the importance of tobacco smoke, wood smoke, and pollution from the ambient environment to concentrations found inside the home.

More extensive research is also needed to understand the biological mechanism driving the exposure-disease relationship. This understanding will help determine not only mechanisms for initial disease onset, but the potential for HAP exposure to lead to negative outcomes among those undergoing TB treatment.

Most importantly, future research should focus on intervention strategies that will successfully decrease exposure to air pollution and the unique settings of low-income urban communities. In addition to the concern about a positive association with TB, these densely populated neighborhoods have high exposures to air pollutants, which may be especially important considering the host of other exposures this population is likely exposed to. For an intervention to be successful, strategies at the neighborhood level will have to be employed given the high prevalence of wood as a secondary fuel source in this community. The proportion of participants reporting exposure to other people burning wood, as well as other neighbors preparing mishri tobacco product, supports this concern. Intervention studies coupled with case-finding strategies may also inform the utility of intervention strategies for household contacts of those diagnosed with pulmonary TB. Household interventions may be strengthened with the complement of additional strategies to increase the welfare of the household, such as nutritional supplementation, which should be investigated.

References

1. The Stop TB Partnership**.** The Global Plan to Stop TB: Update for 2011 - 2015. Geneva; 2010.
2. World Health Organization**.** Global Tuberculosis Report 2015. Geneva; 2015.
3. Lonnroth K, Jaramillo E, Williams BG, et al. Drivers of tuberculosis epidemics: the role of risk factors and social determinants. Soc Sci Med. 2009;68(12):2240-6.
4. Lonnroth K, Castro KG, Chakaya JM, et al. Tuberculosis control and elimination 2010-50: cure, care, and social development. Lancet. 2010;375(9728):1814-29.
5. World Health Organization**.** The Stop TB Strategy. Geneva; 2006.
6. Lim SS, Vos T, Flaxman AD, et al. A comparative risk assessment of burden of disease and injury attributable to 67 risk factors and risk factor clusters in 21 regions, 1990-2010: a systematic analysis for the Global Burden of Disease Study 2010. Lancet. 2012;380(9859):2224-60.
7. Slama K, Chiang CY, Enarson DA, et al. Tobacco and tuberculosis: a qualitative systematic review and meta-analysis. Int J Tuberc Lung Dis. 2007;11(10):1049-61.
8. Kelly FJ, Fussell JC**.** Air pollution and airway disease. Clin Exp Allergy. 2011;41(8):1059-71.
9. Lam NL, Smith KR, Gauthier A, et al. Kerosene: a review of household uses and their hazards in low- and middle-income countries. J Toxicol Environ Health B Crit Rev. 2012;15(6):396-432.

CURRICULUM VITAE
Jessica L Elf, MPH

Curriculum Vitae

Jessica Elf, MPH
Johns Hopkins Bloomberg School of Public Health
Department of International Health
Email: jelf1@jhu.edu
D.O.B: June 5, 1982 (Tallahassee, FL, USA)

EDUCATION

The Johns Hopkins Bloomberg School of Public Health, Baltimore, Maryland

PhD Student, Department of International Health, Global Disease Epidemiology and Control Program

Expected Graduation: December 2015

The Johns Hopkins Bloomberg School of Public Health, Baltimore, Maryland

Masters of Public Health, May 2009

Coursework: Epidemiology, Biostatistics, Program Evaluation

University of Florida, Gainesville, Florida

Bachelor of Science in Nutrition, Cum Laude, April 2004

PROFESSIONAL EXPERIENCE

Institute for Global Tobacco Control, Baltimore, Maryland June 2009 – August 2011

The Johns Hopkins Bloomberg School of Public Health

Research Program Manager

- Development and implementation of research projects designed to support programming and policy decisions related to global tobacco control.
- Development, coordination, and implementation of in-country trainings for data collection, analysis of data and preparation of reports and peer-reviewed publications.
- Development and implementation of activities for the capacity building of students and global tobacco control professionals, including academic curriculum, training activities, and engagement of students and junior researchers and professionals in partnerships with the Institute.

Alachua County Health Department, Gainesville, Florida December 2006 – May 2008

Tobacco Program Specialist

- Responsible for the coordination of county-level tobacco prevention and control efforts at the Alachua County Health Department. Served as a liaison between the Health Department and the Program at the State level.
- Implementation of evidence based tobacco prevention and control programs, established the youth prevention program, SWAT (Students Working Against Tobacco) in middle and high schools, created a tobacco cessation curriculum and facilitated monthly classes, and developed second-hand-smoke education programs.
- Coordinated the establishment of a county Tobacco Prevention and Control Partnership in order to help facilitate policy changes related to tobacco use.

Peace Corps East Timor, Mehara, East Timor July 2005 – May 2006*

Rural Health Promotion Specialist

*Tour ended upon evacuation due to safety concerns by U.S. Government. Program dissolved.

- Assessed healthcare needs in three villages, identifying priorities and serving as a resource and facilitator to local and international health professionals.
- Developed and implemented health promotion activities based upon assessment findings. Facilitated monthly classes educating more than 50 pregnant women and mothers about maternal and child health.
- Collaborated with a local non-governmental organization and community development worker to organize a workshop that would teach young women to construct stoves for their kitchen fires using natural materials, preventing smoke inhalation.
- Elected to and served on the Volunteer Advisory Committee as Chairperson and Treasurer.

University of Florida, Gainesville, Florida May 2004 – May 2005

College of Medicine

Department of Pathology, Immunology, and Laboratory Medicine

Lab Manager

- Designed and implemented experimental procedures on laboratory tissues.
- Supervised the organization, set-up, and maintenance of mouse colonies and cell cultures.
- Trained and managed laboratory personnel on both safety and experimental techniques.

AWARDS AND HONORS

Awards

2014	Ujala Foundation Scholar The Center for Clinical Global Health Education (CCGHE) Johns Hopkins Medicine
February 2013 – May 2014	UJMT Fogarty Global Health Fellow The Johns Hopkins Bloomberg School of Public Health
2014	Student Assembly Student Conference Fund Award The Johns Hopkins Bloomberg School of Public Health
2013	Global Health Establish Field Placement Conference Grant Award "Exposure to indoor air pollution from secondhand tobacco smoke and use of biomass fuels in homes with active TB disease in Klerksdorp, South Africa." The Johns Hopkins Bloomberg School of Public Health Center for Global Health
2012	Global Health Established Field Placement Award "South Africa – Integration of Environmental Assessments into a Mass Active Case Finding Program" The Johns Hopkins Bloomberg School of Public Health Center for Global Health
2008	Master of Public Health Field Experience Award The Johns Hopkins Bloomberg School of Public Health
2000 – 2004	Bright Futures Scholar *(full tuition support for undergraduate education)*

Honors

2004	University of Florida, College of Agriculture and Life Sciences Honors Program
2003	Junior Investigator Travel Award for Poster Presentation Immunology 2003 International Conference Denver, Colorado
2003	Outstanding Basic Science Poster Presentation (co-author) University of Florida, College of Medicine Celebration of Research Day Gainesville, Florida
2002	Phi Kappa Phi Honors Society
2001	Golden Key National Honors Society

PROFESSIONAL ACTIVITIES

Classroom Instruction

2012-2015	340.612.81 Epidemiologic Basis for Tuberculosis Control Johns Hopkins Bloomberg School of Public Health *Teaching Assistant*
2013 – 2014	Sample Size Calculation Lecture ICHORTA AIDS/TB research capacity-building program Johns Hopkins University School of Medicine *Lecturer*
2011 – 2012	140.621.11 – 140.623.11 Statistical Methods in Public Health I – III Johns Hopkins Bloomberg School of Public Health *Tutor*
2010 - 2011	340.662.11 Epidemiology of Tobacco Control Johns Hopkins Bloomberg School of Public Health *Course Coordinator and Teaching Assistant*
2009 – 2010	410.640.81 Global Tobacco Control Johns Hopkins Bloomberg School of Public Health *Course Coordinator and Teaching Assistant*
2009	Tobacco Control Johns Hopkins Bloomberg School of Public Health Johns Hopkins School of Medicine *Teaching Assistant*
2009	410.641.11 Advanced Methods in Global Tobacco Control

Johns Hopkins Bloomberg School of Public Health

Guest Lecturer

Training

2013 Data Collection Workshop

Assessing the association of indoor air pollution with tuberculosis in children under five.

India

Organizer and Trainer

2012 Data Collection Workshop

Integration of Environmental Assessments into a Mass Active Case Finding Program, South Africa

Organizer and Trainer

2011 Data Collection Workshop

Tobacco sales and marketing at the point of sale in Guam.

Organizer and Trainer

2010 Data Collection Workshop

Nicotine Exposure in Children and Families of Tobacco Farming Households in Brazil.

Co-organizer and Co-trainer

2010 Data Collection Workshop

Tobacco Sales and Marketing around Schools in Ahmedabad City, India

Organizer and Trainer

2009 Data Collection Workshop

Point of Purchase Surveillance in 3 Cities in Vietnam.

Co-organizer and Co-trainer

Workgroups, Coalitions, and Partnerships

2010 Johns Hopkins University/American Legacy Foundation Training Grant Working Group

Johns Hopkins School of Public Health

Co-organizer

2008 Alachua County Tobacco Prevention and Control Partnership

Gainesville, Florida

Co-organizer and Member

2006-2008 United Way Fundraising Committee

Alachua County Health Department

Gainesville, Florida

Committee Member

2007-2008 State of Florida Tobacco Disparities Strategic Planning Workgroup

Workgroup Member

2007-2008 Tobacco Prevention and Control Task Force

University of Florida GatorWell Healthy Gator 2010

Gainesville, Florida

Workgroup Member

2007-2008 Partners in Prevention of Substance Abuse (PIPSA) Coalition

CDS Family & Behavioral Health Services, Inc

Gainesville, Florida

Coalition Member

Peer Review

2011-2015 Abstract Reviewer

American Public Health Association

Alcohol, Tobacco, and Other Drugs

2011 Second Level Reviewer

American Public Health Association

Alcohol, Tobacco, and Other Drugs

2012 Abstract Reviewer

SKILLS AND TECHNICAL CAPACITY

- Proficiency in Microsoft Office Suite, Stata, and R programs
- Working knowledge of ArcGIS (ESRI) and PenDragon Forms Programs
- Language: Reading, writing, and speaking proficiency in Tetun language.

PUBLICATIONS

Journal Articles

Elf JL, Modi B, Stillman F, Dave P, Apelberg BJ. Tobacco sales and marketing within 100 yards of schools in Ahmedabad City, India. Public Health. 2013;127(5):442-448.

Avila-Tang, E., **Elf, JL**., Cummings, KM., Fong, G., Hovell, M., Klein, J., McMillen, R., Winickoff, J., Samet, JM. Assessing Secondhand Smoke Exposure with Reported Measures. Tobacco Control. 2013;22(3):156-163.

Clattenberg E, **Elf JL**, Apelberg B. Unplanned Cigarette Purchases and Tobacco Point of Sale Advertising: A Potential Barrier to Smoking Cessation. Tobacco Control. 2013;22(6):376-381.

Wilson, L., Avila-Tang, E., Chander, G., Hutton, HH., Odelola, O., **Elf, JL**, Heckman-Stoddard, B., Bass, EB., Little, E., Haberl, E., and Apelberg, B. Impact of tobacco control interventions on smoking initiation, cessation, and prevalence: a systematic review. J Environ Public Health. 2012;2012:961724 (epub).

Andreis M, **Elf JL**, Johns P, Carvalho A, Yuan J, Apelberg B. Air Quality in Bars of Sao Paulo, Brazil Before and after the Smoke-free Law in Indoor Places. Revista Brasileira de Cancerologia. 2011;57(3): 315-320.

Litherland, S. A., K. M. Grebe, N. S. Belkin, E. Paek, **J. Elf**, M. Atkinson, L. Morel, M. J. Clare-Salzler, and M. McDuffie. Nonobese diabetic mouse congenic analysis reveals chromosome 11 locus contributing to diabetes susceptibility, macrophage STAT5 dysfunction, and granulocye-macrophage colony-stimulating factor overproduction. J. Immunol. 2005, Oct 1; 175(7): 4561-5.

Litherland, S.A., Xie, T.X., Grebe, K.M., Davoodi-Semiromi, A., **Elf J**., Belkin, N.S., Moldawer, L.L., Dysfunction in autoimmune monocytes and macrophages. J. Autoimmun. 2005 Jun; 24(4):297-310.

Oral Presentations

Elf JL, Kinikar A, Khadse S, Mave V, Gupte N, Kulkarni V, Patekar S, Raichur P, Breysse P, Gupta A, Golub J. The association of exposure to air pollution from biomass fuels, kerosene,

and secondhand tobacco smoke with TB in adult women and children in Pune, India. American Thoracic Society International Conference 2015. Denver, Colorado, USA. May 2015.

David A, **Elf JL**, Mummert A, Tamplin SA, Stillman, FA, and Community Research and Action Team, Guam. Using a community-based participatory approach to mapping tobacco point of sale advertising. 15th World Conference on Tobacco or Health (WCTOH). Proffered Paper Sessions: Oral Presentations. Singapore. March, 2012.

Posters

Ogale YP, **Elf JL**, Lokhande R, Mave V, Roy S, Gupta A, Golub JE, Mathad J. Characteristics associated with mobile phone access among tuberculosis patients in Pune, India. 46th World Conference on Lung Health of the International Union Against Tuberculosis and Lung Disease. Cape Town, South Africa. December 2015.

Vaidya A, Tambe S, Bhosale R, Mave V, **Elf JL**, Pradhan N, Nevrekar N, Patil S, Kagal A, Joshi S, Chandanwale A, Gupta A, Mathad J. Operational challenges undermine WHO TB symptom screen in pregnant women. ID Week. San Diego, California, USA. October 2015.

Elf JL, Eke O, Rakgokong MH, Variava E, Baliram Y, Lebina L, Shapiro AE, Breysse P, Golub J, Martinson N. Exposure to indoor air pollution from secondhand tobacco smoke and use of biomass fuels in homes with active TB disease in Klerksdorp, South Africa. 44th World Conference on Lung Health of the International Union Against Tuberculosis and Lung Disease. Paris, France. November 2013.

Khan A, Khan A, **Elf JL**, Golub J. Prevalence of tobacco smoking among newly diagnosted pulmonary tuberculosis patients attending DOTS clinics in Karachi, Pakistan. 43rd World Conference on Lung Health of the International Union Against Tuberculosis and Lung Disease. Kuala Lumpur, Malaysia. November 2012.

Elf, JL., Almeida, G., Avila-Tang, E. Nicotine exposure and symptoms of green tobacco sickness (GTS) among children and members of tobacco farming households in Brazil. 15th World Conference on Tobacco or Health (WCTOH). Singapore. March 2012.

Clattenberg E, **Elf JL**. Unplanned Cigarette Purchases and Tobacco Point of Sale Advertising: A barrier to reducing cigarette smoking. American Medical Student Association Annual Convention. Washington DC. March 2012.

Modi, B., Stillman, F., **Elf, J.**, Apelberg, B., Breyssee, P., Dave, P., Parmar, G. Tobacco Sales and Marketing around Schools in Ahmedabad City, India. Asia Pacific Conference on Tobacco or Health. Sydney, Australia. October 2010.

Stillman, F., Kibria, N., **Elf, J.**, Giang, K.B., Breysee, P., Madeiros, D. Point of Purchase Surveillance in 3 Cities in Vietnam. Asia Pacific Conference on Tobacco or Health. Sydney, Australia. October 2010.

Elf, J., Meyers, M., Almeida, L.M., Apelberg, B., Figueiredo, V., Hepp, L., Avila-Tang, E. Assessment of Second-Hand Smoke (SHS) Exposure and Point of Purchase (POP) Tobacco

Advertising in Rio de Janeiro, Brazil. Conference of the American Public Health Association. Philadelphia, PA, USA. November 2009.

Elf, J., N. Belkin, B. Rumore-Maton, J. Elf, N. Belkin, M. J. Clare-Salzler, M. McDuffie, and S.A. Litherland. 2004. STAT5 Dysregulation in Autoimmune Type 1 Diabetes(T1D). for the12th International Congress of Immunology and 4th Annual Conference of FOCIS, Montreal, Canada, July 2004.

Elf, J., N. Belkin, B. Rumore-Maton, J. Elf, N. Belkin, M. J. Clare-Salzler, M. McDuffie, and S.A. Litherland. 2004. STAT5 Dysregulation in Autoimmune Type 1 Diabetes(T1D). Abstract for UF College of Medicine Research Day. April 2004.

Litherland, S. A., B. Rumore-Maton, **J. Elf**, N. Belkin, M. J. Clare-Salzler, and M. McDuffie. 2004. GM-CSF, PGS2, & STAT5 Dysregulation in Autoimmune Monocytes & Macrophages. for Cold Spring Harbor Meeting: Gene Expression & Signaling in the Immune System 4/28-5/2/04.

Litherland, S. A., B. Rumore-Maton, **J. Elf**, N. Belkin, M. J. Clare-Salzler. 2004. STAT5 Dysfunction in Autoimmune Monocytes & Macrophages. For the Keystone Symposium: Jaks and STATs, Whistler, British Columbia, Canada, April 15-20, 2004.

Litherland, S.A., **J. Elf**, E. Paek, M.J. Clare-Salzler, and M. McDuffie. Abstract for AAI Annual Meeting, Denver, Colorado. Myeloid Cell GM-CSF, STAT5, and Prostaglandin Synthase 2 (PGS2/COX2) Expression in Nonobese Diabetic (NOD) Mice Myeloid Cell GM-CSF, STAT5, and Prostaglandin Synthase 2 (PGS2/COX2) Expression in Nonobese Diabetic (NOD) Mice. Awarded AAI Junior Faculty Travel Award. May 2003.

Litherland, S.A., **J. Elf**, E. Paek, W. -K. Tse, R. Peng, K L. Womer, A. Davoodi-Semironi, Y. Li, V. Dharnidharka, M.J. Clare-Salzler, L. Morel, and M. McDuffie. Abstract for UF College of Medicine Research Day. STAT5 Dysfunction in Autoimmune Myeloid Cells Activation and Differentiation. Awarded Best Poster Presentation-Basic Sciences. April 2003.

Litherland, S.A., **J. Elf**, E. Paek, W. -K. Tse, R. Peng, K L. Womer, A. Davoodi-Semironi, Y. Li, V. Dharnidharka, M.J. Clare-Salzler, L. Morel, and M. McDuffie. Abstract for Dendritic Cell Keystone Symposium. STAT5 Dysfunction in Autoimmune Myeloid Cells Activation and Differentiation. Awarded Invitation for Workshop Oral Presentation. March 2003.

Litherland, S.A., **J. Elf**, Y. Li, R.H. Peng, M. McDuffie, and M. J. Clare-Salzler. Abstract for Salk Institute Tyrosine Phosphorylation and Signal Transduction Meeting 2002. STAT5 Dysfunction in Type 1 Diabetes : Persistent Tyrosine Phosphorylation, Aberrant DNA Binding, & Altered Subcellular Translocation During Myeloid Cell Differentiation. July 2002.

Book Chapters

Avila-Tang E, **Elf J**. Tobacco use and disease, epidemiology of. In: Rippe JM. Ed.: Encyclopedia of Lifestyle Medicine and Health. Thousand Oaks, CA: SAGE Publishing, 2012.